ORAL HISTOPATHOLOGY

A MANUAL FOR STUDENTS AND PRACTITIONERS OF DENTISTRY

BY

MARTIN A RUSHTON
M.A M D., Odont D., F D.S.R C.S.Eng & Edin
Professor of Dental Medicine University of London

AND

BRIAN E D COOKE
M R C.S., L.R C.P., F D.S R C.S Eng
Reader in Dental Medicine University of London



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PREFACE

It is possible in a small book to show and to relate what is happening at a cellular level in the principal disorders of the teeth and neighbouring parts and that is our intention To explain in detail why these things are happening so far as we know this would require much further discussion of environmental factors and alternative hypotheses than it is the aim of this book to offer It is hoped however that a limited factual presentation will not only assist students and practitioners to comprehend the nature of the changes in the tissues they observe and treat but will in many arouse the natural desire for a more complete and satisfactory explanation of them than can be provided here or may yet be available The brief bibliographies offer suggestions for wider reading or indicate the source of photomicrographs previously published They do not include all the best articles on any aspect but offer starting points for further enquiry That they are almost confined to papers in the English language must not be taken as a reflexion on the value of original work published in other languages but be understood as intended for the convenience of readers using the English tongue.

No authors of a text book of oral histopathology can be without obligation to the late Dr R A Kronfeld and to Dr K H Thoma It is a pleasure to express our indebtedness to them and to the leaders of dental research in every land

We wish to express our particular thanks to Mr J E. Hutchinson who has made nearly all the histological preparations and photomicrographs used here to the Editors of the *British Dental Journal* *Oral Surgery Oral Medicine and Oral Pathology* the *Journal of Pathology and Bacteriology* the *Annals of the Royal College of Surgeons of England* and the *British Journal of Dermatology* who have allowed us to reproduce illustrations used by us in previous papers of which they hold the copyright to colleagues who have lent us sections of pathological material or allowed us to reproduce figures from their work particularly Professors R V Bradlaw and A I Darling Drs G Gustafson and J L. Hardwick Professors R B Lucas and E. B Manley and to Miss N Carlton for help in preparation of the manuscript. We are also greatly obliged to Messrs E & S Livingstone for their courtesy and assistance

M A RUSHTON
B E. D COOKE

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I

TEETH AND THEIR FORMATIVE ELEMENTS

1 DEVELOPMENTAL ANOMALIES

Developmental anomalies are common and range from minor features scarcely outside normal variation to gross defects as regards number morphology and structure. The anomalies to be described are those in which defects of structure are important

STRUCTURAL DEFECTS OF ENAMEL

Genetically Determined Amelogenesis Imperfecta. Several forms of structural defect of enamel have a familial incidence and are thought



FIG 1
Hereditary enamel hypoplasia a moderately severe (female) case showing vertical wrinkles.

to result from genes which are inherited These disorders are usually divided into two groups (Weinmann *et al* 1945) hereditary enamel hypoplasia and hereditary enamel hypocalcification Somewhat intermediate forms are also found (Witkop 1958) Usually all teeth are affected, though not always equally

HEREDITARY ENAMEL HYPOPLASIA. Here the enamel is hard but too thin, so that in extreme cases the teeth appear almost denuded of enamel In less severe cases the surface is pitted or wrinkled in vertical grooves (Fig. 1) The teeth suffer quickly from attrition In ground

sections it may be seen that the quality of the enamel is abnormal: while some areas show the usual rod structure, others have a glassy appearance with fine laminations parallel with the surface (Fig 2). On decalcification these areas leave a matrix of hyaline, structureless appearance

HEREDITARY ENAMEL HYPOCALCIFICATION. In these cases the enamel is normal in quantity but incompletely calcified so that it is unduly soft and has a matt surface After eruption the enamel becomes pigmented buff, orange or brown, and may be quickly chipped and worn

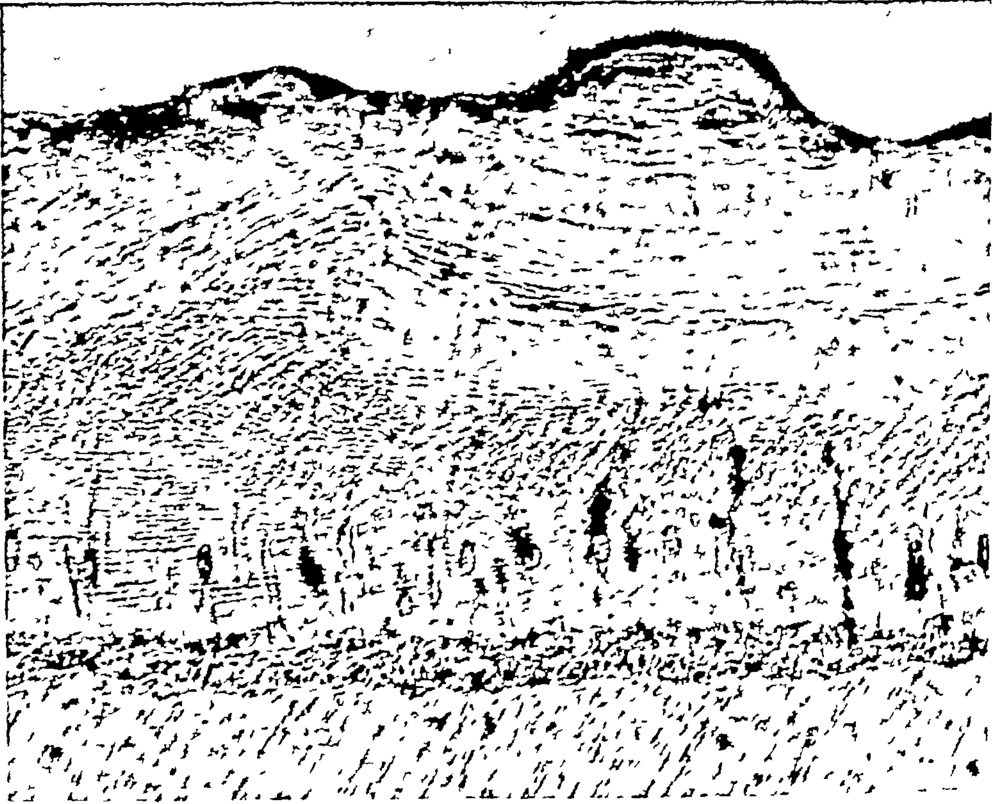


FIG 2

Hereditary enamel hypoplasia ground section from a severe (female) case showing thin enamel, of which the superficial layers are of glassy appearance without visible rod structure ×500

away Much of the enamel matrix remains acid-insoluble, as in incomplete maturation, so that it is often preserved in sections of decalcified specimens The rod pattern of the enamel is approximately normal Sections of young unerupted teeth show premature degeneration of the enamel organ (Fig 3)

Metabolic Disorders and General Infections. Disorders of calcium metabolism such as rickets and hypoparathyroidism, if occurring during the period of tooth formation, are likely to result in hypoplastic defects

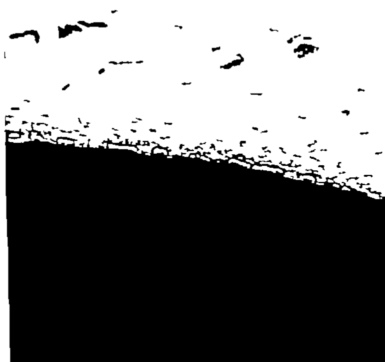


Fig. 3

Hereditary enamel hypocalcification on a child's unerupted premolar the reduced enamel epithelium has already degenerated, leaving only some hyaline nodules on the surface of the enamel From a section kindly lent by Professor E. B Manley Van Gieson × 60



Fig 4

Enamel hypoplasia associated with tetanic convulsions which occurred at two years of age

of the enamel formed at the time of illness (Fig 4) They will also cause defective calcification not macroscopically visible

Severe disturbances of health such as may occur in the first weeks of life of a premature infant, or exanthematous fevers in childhood may result in hypoplastic pits and grooves in the enamel, usually arranged horizontally In some cases the cause may be insufficient availability of mineral substances, in others toxic damage to the enamel-forming cells

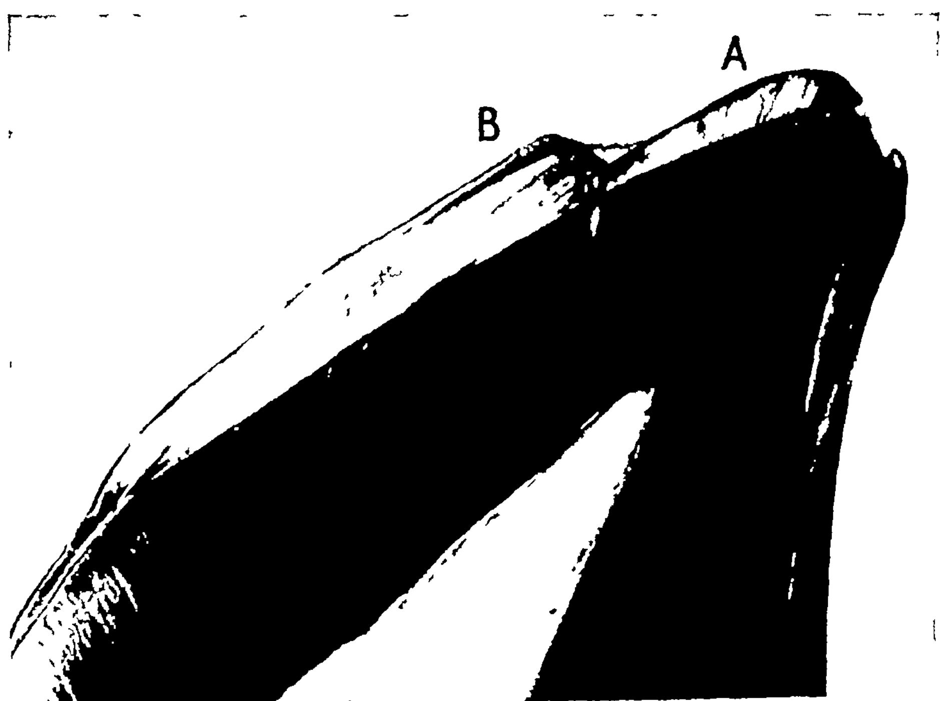


FIG 5

Neonatal hypoplasia of enamel Ground section (×18) of an upper lateral deciduous incisor of a child born at the 28th week of pregnancy There is a notch in the labial enamel At A the enamel matrix formed before birth has never been covered by the enamel which should have been formed over it after birth At B the prenatal enamel is covered by a normal thickness deposited post-natally superficial to the neonatal line

The appearance of a ground section of a deciduous tooth with *neonatal hypoplasia* is characteristic The neonatal line indicates the level reached by enamel matrix formation at the time of birth In neonatal hypoplasia there will at some parts be no enamel lying superficial to this line so that the prenatal enamel is exposed on the surface of the tooth (Fig 5) Nearer to the neck of the tooth the formation of enamel has been resumed This means that some of the ameloblasts active at the time of the disturbance—sometimes all of them—perished, but that the younger ameloblasts which had not yet begun to produce enamel matrix escaped

A ground section of a *rachitic* tooth shows depressions on the enamel surface corresponding to the grooves and pits seen macroscopically (Fig. 6) The contour lines of the enamel have the appearance of having collapsed together under these depressions and it has been suggested that a collapse of poorly calcified enamel matrix has in



FIG. 6
Ground section of human rachitic tooth Two deep grooves seen on the enamel surface correspond to periods of disturbed enamel formation. Interglobular dentine (best seen opposite the lower groove) was formed at the same periods. × 30

fact occurred However a reduced rate of matrix formation by the ameloblasts in the affected areas would give the same appearance The disturbances in the enamel *can usually be seen to correspond* chronologically with areas of interglobular dentine in the same specimen

The appearance of tooth germs in human rickets has been described by Gottlieb (1920) There are degenerative changes in the enamel organ which may be partly separated from the enamel surface by an exudate containing cells Ameloblasts may no longer be recognised

in some areas, and the enamel matrix varies in thickness from one part to another or may in some places have failed to form at all Most information about the effects of vitamin D deficiency has been derived from experiments on animals In human rickets vitamin D deficiency is



FIG 7
Tooth germ of Hutchinsonian incisor showing abnormal outline of the dentine papilla with ridges and central depression (left) which will correspond to the incisive notch Haematoxylin and eosin (Bradlaw, 1953 *Oral Surg* **6**, 147)

often only one of several deficiencies concerned in the illness and the results may differ from those described in animal experiments

Less severe disturbances than those necessary to produce macroscopic defects of the enamel will cause marked incremental lines visible in ground sections, or areas of defective mineralisation which can be recognised more certainly by other physical methods than simple optical examination, for example by radiographs of sections or by the polarizing microscope

Prenatal Syphilis causes the characteristic deformity of the permanent upper central incisors (Hutchinsonian teeth) and of the first permanent molars The abnormal shape reflects changes in the odontogenic epithelium (Fig 7). According to Bradlaw (1953) perivascular infiltration and oedema of the follicle are followed by hyperplasia of the external enamel epithelium stratum intermedium and ameloblasts



FIG. 8
Stained ground section of a permanent tooth damaged during formation by an infective process (osteomyelitis of the jaw). The irregular surface of the enamel E is covered with cementum C. The dentine D has remained thin and was only calcified at its outer surface, the predentine remaining very wide and here stained dark. The pulp P has pulled away from this slightly in preparation Picrothionin ×70

which bulge into the dental papilla producing the characteristic notch and an irregular amelodentinal junction. Enamel is defectively formed and may in parts be entirely lacking and dentine is often hypoplastic. By appropriate methods *Treponema pallida* may be found throughout the tooth germs.

Fluorosis Where the fluoride content of water habitually drunk in childhood much exceeds one part per million the structure of the

enamel formed during that period is altered in such a way that visible effects are produced These effects vary from opaque white patches to irregular brown areas, producing the characteristic 'mottled enamel' Fluorosis is, however, not the only cause of opaque white patches When the fluorosis is moderate, the enamel retains a fine surface gloss, but when severe, the surface may be dull and pitted and the enamel may have lost its usual strength In ground sections the affected enamel appears defectively calcified both as regards the rods and interprismatic material The rods may be sharply outlined by a brown substance lying between them

Local Infection and Trauma. Hypoplastic defects of the enamel of single teeth may result from infection about the roots of a deciduous predecessor or mechanical injury to the developing tooth (see also page 69) In the case of infection around a tooth germ, the enamel may be missing over a limited area due to local destruction of the enamel organ or may be thin and irregular Defects in its surface are often partly filled up by apposition of cementum before the tooth erupts (Fig 8) In severe cases such as in osteomyelitis of the jaw, the apposition of dentine may also be permanently arrested so that a stunted tooth is formed The quality of dentine may also be most abnormal, resembling that formed during rickets

STRUCTURAL DEFECTS OF DENTINE

Genetically Determined : Hereditary Opalescent Dentine. A disorder due to an inherited dominant gene, and affecting all the teeth of both dentitions in most cases, is known as hereditary opalescent dentine or dentinogenesis imperfecta A similar anomaly occurs in some patients with the bone disorder osteogenesis imperfecta The teeth tend to be small with bulbous crowns and small roots (Figs 9, 10, 11), are somewhat translucent on eruption and later become gradually grey or brown with bluish reflections from the enamel The teeth wear away quickly The disorder is principally in the formation of the dentine, but the enamel is often poorly calcified and tends to break away and become lost early in some cases

The dentine near the amelo-dentinal junction is usually normal but that lying more deeply shows disordered structure with a diminished number of tubules, poor calcification, imperfect formation of the collagenous matrix and marked irregular incremental lines (Figs 12, 13) The pulp cavity becomes obliterated early and there may be numerous pulp stones included in the dentine The tubules are very irregular in size and in their course It appears that odontoblasts are normally differentiated, but soon degenerate and are then replaced by

others which also disappear and are again replaced Patches of more normal dentine often occur beneath the cusps and sometimes in the whole crown Small blood vessels and cells may be included in the



FIG. 9
Hereditary opalescent dentine Radiograph shows a first permanent molar with bulbous crown and small roots Although the tooth has just erupted at seven years, the pulp cavity is obliterated. (*Ann roy Coll Surg Engl* 1955)



FIG. 10
The deciduous teeth are brown and worn to the level of the gum The permanent incisors are beginning to change colour (*Ann roy Coll Surg Engl* 1955)

dentine. In the deciduous teeth sometimes the pulp cavity is not obliterated, but the dentine on the contrary remains thin and the pulps may be exposed by attrition.

Rather similar conditions, which may be variants of hereditary opalescent dentine, are known, in which there is only a thin shell of dentine in the permanent teeth or where the roots are very short and composed of a mass of embedded pulp stones

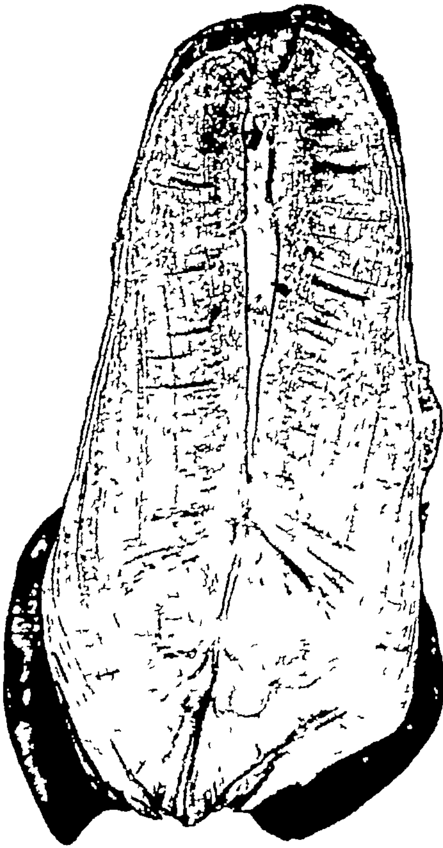


FIG 11

Ground section of upper central incisor from a patient with osteogenesis imperfecta Note stumpy shape, obliteration of pulp cavity, embedded pulp stones, and radiating vascular inclusions in the dentine ×7

In hereditary vitamin D resistant rickets the dental defects are as in acquired rickets (see below)

Metabolic Disorders and Infections. RICKETS Forming teeth examined during the active stage of rickets show increased width of the predentine layer and incomplete calcification of recent dentine (Fig 14) The completed teeth of those who have suffered from rickets during the period of tooth formation show bands and areas of interglobular dentine corresponding to the periods of illness (Fig 15) and often accompanied by enamel defects While the bands of interglobular dentine lie along the incremental lines, the defect will be more severe at the ends of the lines near the surface of the tooth but not quite reaching the enamel or cementum An impression that the defects lie in a line parallel with the outer surface of the dentine is thus often produced (Fig 16) Other disturbances of calcium metabolism will cause analogous effects Thus in idiopathic *hypoparathyroidism* the dentine will show lines of disturbed calcification corresponding with periodic bouts of tetany (Fig 17) Interglobular dentine is not, however, a prominent feature, perhaps because the rate of growth of the dentine matrix is retarded at the same time that calcification is disturbed, as shown by the stunted root growth

SCURVY In the tooth germ will be found old and recent haemorrhages of the pulp, some of which may be followed by calcification,

rrest of dentine formation due to inability to form collagenous matrix nd degenerative changes in the odontoblasts (Figs 18 19) In the eeth of adults who have had scurvy may be found disturbances in the



FIG. 12
Osteogenesis imperfecta The peripheral dentine is more normal than the deeper layers which contain few tubules and may be deficient in collagenous matrix also Picrothionin. × 160 (*Ann roy Coll Surg Engl* 1955)

pattern of dentinal tubules corresponding to the period of vitamin C deficiency and calcified formations in the pulp The effects of scurvy on the dentinal tissues have been chiefly studied in guinea pigs

ILLNESSES OF GREAT SEVERITY will produce temporary arrest of dentine formation with accentuated incremental lines. In prenatal syphilis the calcification of the dentine may be defective In some



Fig 13

Osteogenesis imperfecta owing to deficiency of collagen the dentine has a threadbare appearance in silver impregnated sections A capillary loop (centre) has been included in the matrix ×160 (*J Path Bact* 1939)



Fig 14

Ground section of incomplete tooth of a rachitic child showing both hard and soft tissues stained with carmine The predentine, shown as a black vertical band on the right, is about three times normal width (From a specimen kindly lent by Lady M Mellanby) ×45



Fig 15

Very poorly calcified dentine formed soon after birth in a second deciduous molar of a child with hereditary vitamin D resistant rickets. Haematoxylin and eosin. × 50



Fig 16

Ground section (× 50) showing interglobular dentine near the periphery of the incremental lines but stopping short of the enamel The general effect is of a layer parallel with the



FIG 17

Hypoparathyroidism Lower second molar with caries of the occlusal surface The dentine shows very marked incremental lines of lighter colour corresponding to periods at which calcification was severely disturbed and the child had tetanic fits Haematoxylin and eosin ×8

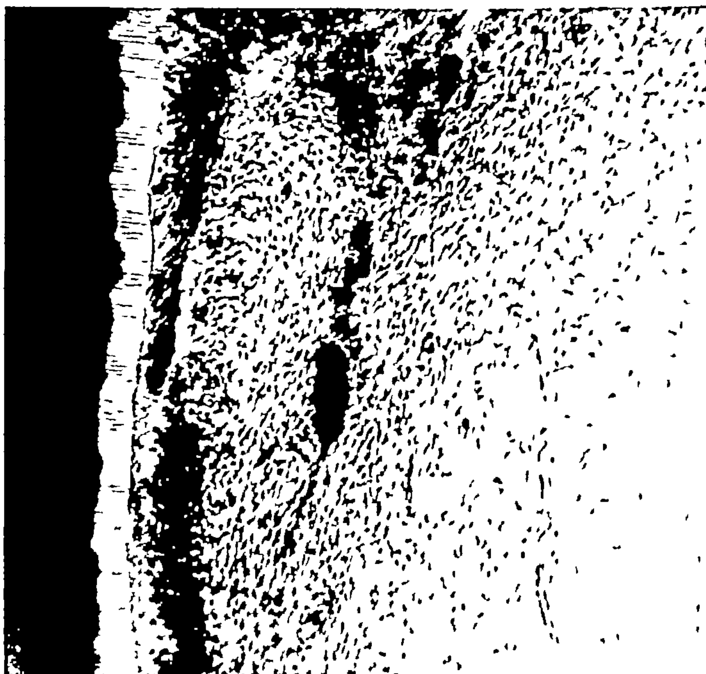


FIG 18

Unerupted premolar germ from a scorbutic child An old haemorrhage in the pulp is partly calcified Haematoxylin and eosin ×90 *(Brit dent J 1950)*

disorders *pigmentation* is a feature Thus in porphyria congenita the dentine is coloured pink or red by the abnormal deposition of porphyrins in its substance in bands of varying intensity In congenital haemolytic



FIG. 19
A rather more recent haemorrhage in the same pulp has stripped up the odontoblast layer Dentine formation has also been arrested Haematoxylin and eosin ×320 (*Brit dent J* 1950)

anaemias there are narrow bands of green pigment in the dentine corresponding to the neonatal period (Fig. 20)

LOCAL INFECTION AND TRAUMA may cause arrest or modified development of the dentine (see pages 8 and 69)

ODONTOMES

While the results of inflammatory hyperplasia and neoplasia of dental tissues have frequently been included under odontomes the term is here applied to developmental anomalies of the dental formative tissues

regarded as outside the range of normal variation They are malformations of unknown origin, possibly the result of some kind of embryological accident, though in a few cases there may be evidence of hereditary predisposition Examples of those types which present features of histological interest will be described

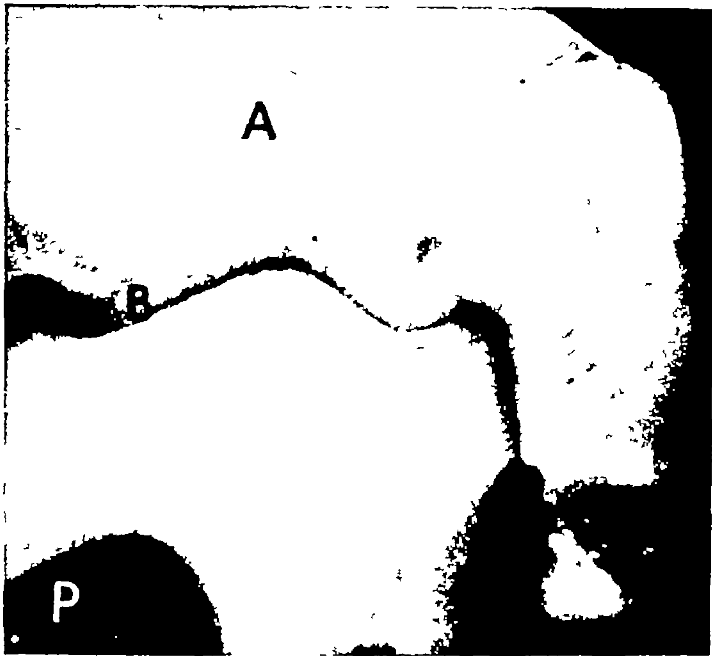


FIG 20
Congenital haemolytic anaemia Second deciduous molar split, polished, and photographed by reflected light Amelodentinal junction at A, band of green pigment corresponding to the neonatal period at B, pulp cavity at P There is a carious cavity at lower right corner ×7

Complex Odontome. These structures have little resemblance to a tooth, though composed of dental tissues They arise in connection with the second dentition and may take the place of one or even two teeth or may be additional structures Their growth is commonly completed at the same age as that of the teeth

The macroscopic appearance of a mature specimen is a spherical or lobulated mass, varying in size from that of a pea to a plum and separated from the surrounding bone by a thin membrane Beneath this is a nodular surface covered by a thin layer of cementum and sometimes portions of enamel The mass is as hard as bone or harder and heavy for its size The cut surface characteristically shows a radiating surface (Fig 21) In sections this is seen to be due to alternating layers or spicules of enamel and dentine with spaces containing tissue homologous with the dental pulp



FIG. 21
Cut and polished surface of a complex odontome showing radiating structure ×2.



FIG 22
Section from a complex odontome occurring in a baby In the part shown the formation of dental hard tissues had not yet begun Disorderly epithelial proliferation is seen.

In specimens removed from infants, the stages of development can be seen (Figs 22, 23) The odontogenic epithelium which normally defines the shape of a tooth here proliferates in a disorderly manner As it still has the property of inducing the differentiation of odontoblasts and the formation of dentine and can itself produce enamel, the result is the apposition of these dental hard tissues in a confused pattern. As adult life is reached, growth ceases and cementum is deposited on the exterior of the mass



FIG 23
In another part of the specimen shown in Figure 22 the apposition of dentine has commenced next to the columnar cells on the surface of the epithelial proliferation (*Brit dent J* 1944)

In ground sections little more can be seen than a confused mixture of the hard dental tissues In sections of decalcified specimens which have not been infected, it is possible to identify various characteristic features A common appearance is a sheet of poorly calcified dentine in which there are many irregular spaces (Figs 24, 25) Some of these contain dental pulp tissue, others decalcified enamel matrix and hyaline material, sometimes with ameloblasts still active, and other parts of an enamel organ with supporting connective tissue The odontome is separated from the surrounding bone by an interval contain-

ing connective tissue epithelial débris, droplets of structureless enamel substance and nodules of cementum When there is infection the epithelial débris will proliferate (Fig. 26) In some specimens no enamel may be formed These are sometimes classified separately as dentinoma

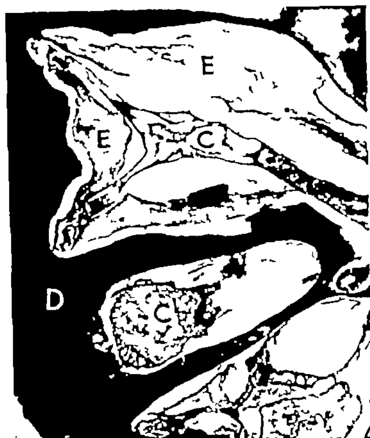


Fig 24

Complex odontome The section shows a sheet of dentine D in which are many spaces containing enamel matrix E. In the middle of the spaces on the surface of the enamel is a reduced enamel epithelium supported by connective tissue C Haematoxylin and eosin ×40

Compound Odontome. These odontomes consist of a number of tiny denticles lying close together in connective tissue and occasionally in the wall of a cyst. Many of the denticles are well formed and in sections of a young specimen a process resembling normal tooth formation is observed Other irregular structures may resemble those seen in a complex odontome. In the connective tissue epithelial débris enamel droplets and cement nodules may be found Growth ceases at maturity

Soft Odontomes. This name has been applied to several *different* proliferations including the adamantinoma and some fibromata which

are neoplasms, and the immature complex odontome It is here used to indicate those malformations of the dental formative organs in which there is hyperplasia of fibrous connective tissue containing proliferations of odontogenic epithelium, but with only inconsiderable traces



FIG 25
Complex odontome The white spaces were occupied before decalcification by enamel and remnants of enamel matrix can be seen At lower left a regular layer of columnar ameloblasts covers the enamel surface In the centre are many globular deposits of hyaline enamel among epithelial débris, and connective tissue being replaced by a calcified matrix Haematoxylin and eosin × 120

of enamel, dentine or cementum (Fig 27) The growth of these swellings is generally limited to the developmental period, but similar structures are occasionally reported as continuing to grow in adult life and have been regarded as neoplastic in those cases

Invaginated Odontome. (*Dens in Dente, Dilated Odontome*) This is a malformation of the tooth in which during development the



FIG. 26
Complex odontome The epithelial débris in the fibrous capsule have proliferated P as a result of infection. Irregular areas of enamel E are surrounded by poorly calcified dentine D Haematoxylin and eosin. ×60



FIG 27
Soft odontome Strands of epithelium are scattered in a mass of mature fibrous tissue Some are surrounded by a hyaline margin of collagenous material. Haematoxylin and eosin ×60

enamel organ becomes infolded, so that part of it protrudes into the dentinal papilla As a result there exists within the dentine a cavity communicating with the exterior of the tooth by a narrow passage and lined in part by enamel The pulp cavity of the tooth has often been displaced by this abnormal process and the apex may remain open In some cases the invagination is such that the tooth is greatly expanded (dilated odontome) The point of entry of the invagination can often be identified as a small pit on the surface of the crown, but occasionally it may lie on the root and then usually cannot be identified Some specimens are of great complexity but in a simple type the following can be seen in a section (Figs 28, 29): the point of entry, the invagination cavity containing some enamel on its wall, and in a young unerupted specimen remnants of the enamel organ and its supporting connective tissue (Fig 30), and the dentine surrounding the cavity which usually contains a compressed slit-like pulp The dentine between the floor of the invagination cavity and the pulp is often very poorly formed or incomplete, and the passage of micro-organisms through this imperfect barrier frequently leads to early infection of the pulp

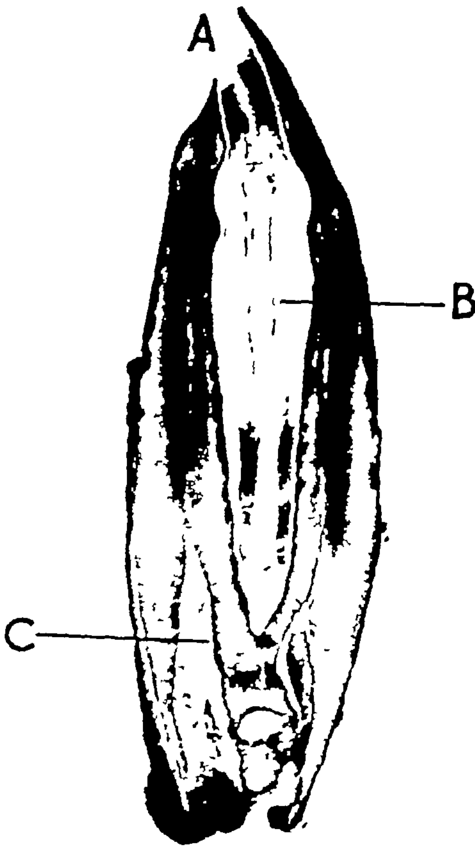


FIG 28

Invaginated odontome The crown of the tooth is uppermost The entrance channel A leads to the invagination cavity B in which are the decalcified remains of an enamel lining The pulp C lies in a narrow slit in the dentine on each side and fills the open apex Haematoxylin and eosin ×5

In specimens which have erupted, the invagination cavity usually contains infected necrotic material from which this bacterial invasion extends

Evaginated Odontome (oriental premolar, axial core odontome) A malformation of premolars and other teeth found in Malaya and Japan is characterized by the presence of an elevated nodule in the middle of the occlusal surface It may be regarded as the result of a developmental extrusion of dental papillary substance into the enamel organ

and to that extent the reverse of an invaginated odontome. The nodule contains a fine extension of the dental pulp As the nodule soon becomes worn down or fractured, the pulp becomes exposed and pulpitis follows

'Geminated' Teeth. These are malformations sometimes hereditary having the appearance of two or sometimes three teeth joined



FIG. 29

An enlarged view of the entrance channel (× 50) shows dentine at extreme right and left The channel has become almost filled with enamel of which the organic matrix is seen

together Histological examination shows that dentine unites the two or more parts (Fig 31) which may at the time of examination have either one common or separate pulp cavities The condition is to be contrasted with that found in concretion false gemination where two separate teeth are united by a deposit of cementum (page 89)

Enamel Nodules and Plaques. In this malformation there is an area of the root covered with enamel in a situation which would be expected

to be covered with cementum In some cases the enamel protrudes as a nodule covering a core of dentine, into which there may be a prolongation of the pulp cavity. In others a tiny disc of enamel lies flat upon the surface of the dentine of the root (Fig 32) This type is extremely

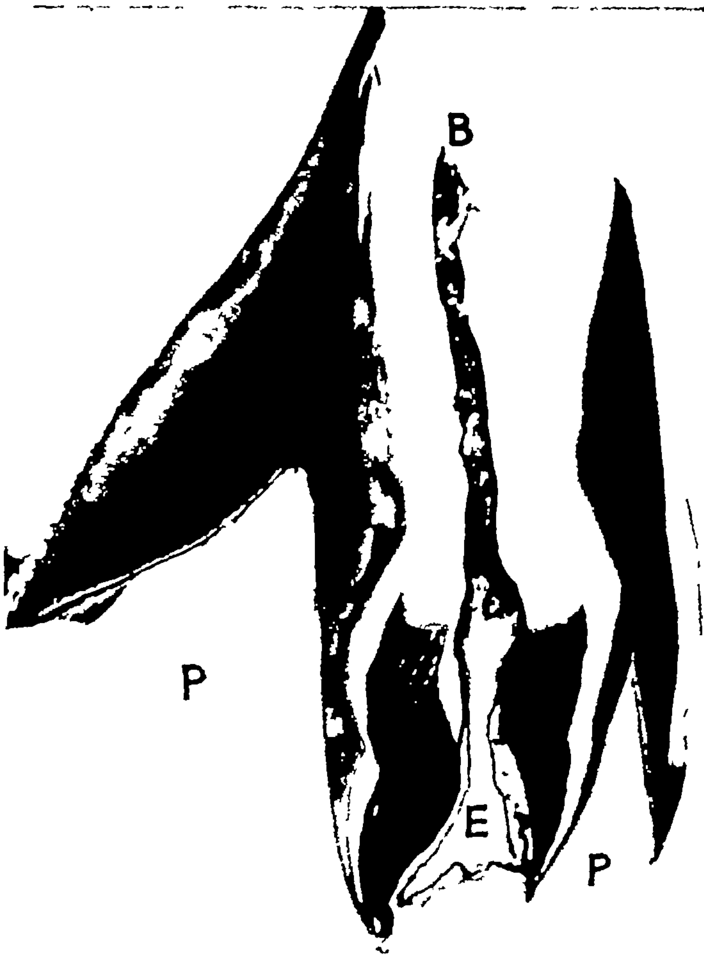


FIG 30
Young invaginated tooth, of which only the superficial part of the crown has yet formed In the centre is the invagination channel containing enamel organ E with a cord of blood vessels B from the follicle The pulp lost at operation, should occupy the lower part of the figure PP Haematoxylin and eosin × 12

common between the roots of molars and the enamel tends to become covered over with cementum in older patients Droplets of hyaline enamel substance are often found in its vicinity in the periodontal membrane In this anomaly some cells of Hertwig's sheath evidently possess the property of forming enamel

Cementoma. Under this name have been described hyperplastic disorders of cementum and bone of several kinds (see page 90)

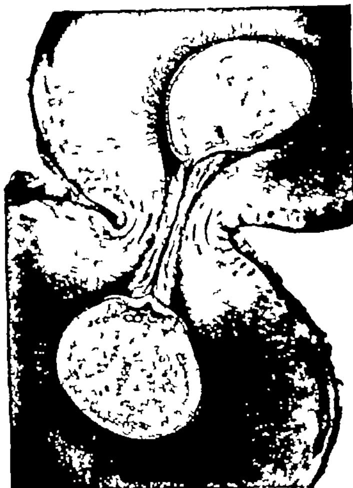


FIG 31
Horizontal section through a geminated tooth There is a bridge of dentine joining what would otherwise be two teeth Picrothionin × 14



FIG. 32
A small disc of enamel of which the organic matrix is visible lies near the bifurcation of the roots. Haematoxylin and eosin. × 75

DEVELOPMENTAL CYSTS, ODONTOGENIC IN ORIGIN

The gingivae and the alveolar portion of the jaws abound with odontogenic epithelium This is actively proliferating during the years of tooth formation, later remains as epithelial rests, and will on occasion form cysts, some of clinical importance, others only identifiable microscopically By far the most important and common of those of developmental origin is the dentigerous cyst which arises from the enamel organ after the crown of the tooth is formed Small dentigerous



FIG 33
Dentigerous cyst Flattened layer of stratified squamous epithelium supported by fibrous tissue free from inflammatory cells Haematoxylin and eosin × 80

cysts situated over the crowns of teeth about to erupt are known as cysts of eruption Should the enamel organ undergo cystic change and fail to produce a tooth, then the cyst is called a primordial cyst The evidence for the enamel organ rather than the dental lamina being the source of the primordial cyst is only circumstantial, such as a cyst developing in place of a permanent tooth Remnants of the dental lamina sometimes undergo cystic change and interfere with the eruption of a deciduous tooth, these cysts are also known as cysts of eruption More often they retain microscopical proportions and are classed as gingival cysts Finally the epithelial rests of Malassez occasionally give rise to cysts known as developmental periodontal cysts, to distinguish

them from the majority of periodontal cysts which are infective in origin.

The Dentigerous Cyst. The dentigerous cyst envelops the whole crown of a tooth or only part of it It is known as the central type if

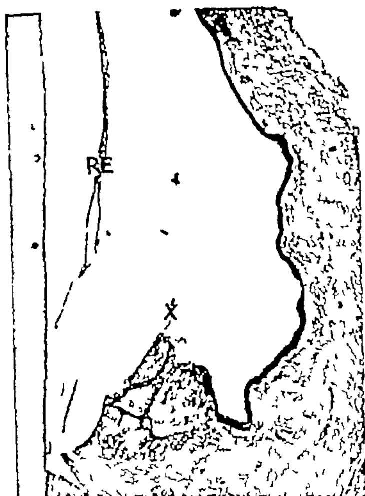


FIG. 34

Dentigerous cyst. The epithelial lining of the cyst merges with the reduced enamel epithelium covering the crown at the amelo-cemental junction In the preparation of the section the enamel has been lost and the reduced enamel epithelium RE has been displaced and torn at X. Haematoxylin and eosin ×60

the crown is completely enclosed in the cyst and the lateral type if it is just related tangentially to it. It is lined by epithelium and contains fluid under pressure

It is a developmental anomaly arising from the enamel organ after amelogenesis has been completed Hypoplastic defects of the enamel that might suggest an earlier breakdown of the stellate reticulum are rarely found, and the enamel is covered with the reduced epithelium.

Teeth involved are often those of which the eruption has been delayed, and multiple dentigerous cysts may be found in the jaws of persons affected by cleido-cranial dysostosis The cysts that arise after the enamel organ has become the reduced enamel epithelium may develop from neighbouring remnants of the dental lamina

The epithelial lining of the dentigerous cyst is a uniformly flattened layer of stratified squamous epithelium, supported by fibrous tissue (Fig 33) Only occasionally is the epithelium keratinized At the

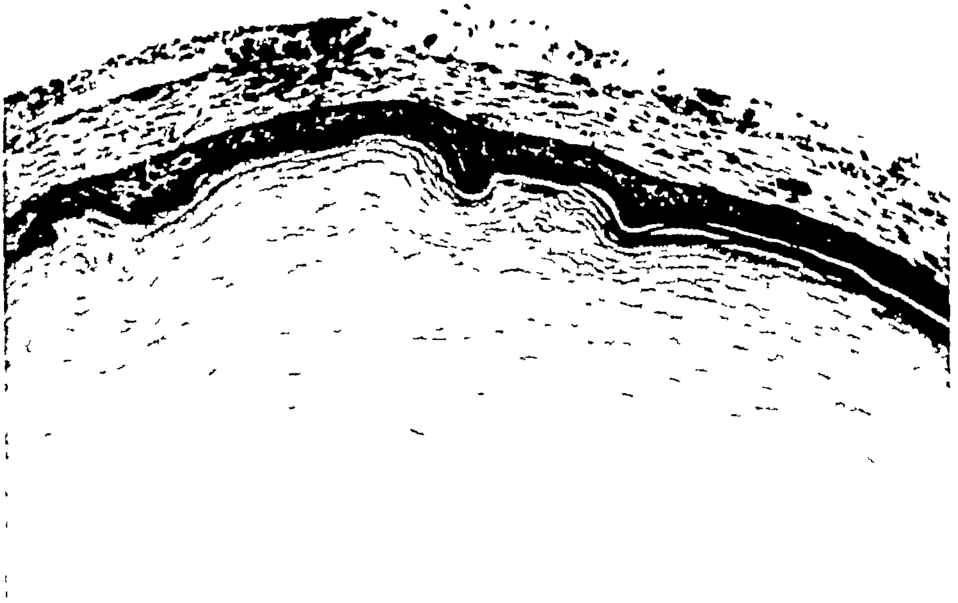


FIG 35

Primordial cyst The epithelial lining is well keratinised Haematoxylin and eosin × 145

amelo-cemental junction the epithelial lining merges with the reduced enamel epithelium covering the crown, and the fibrous tissue with the periodontal membrane (Fig 34) If the cyst has become infected then the cyst wall will be infiltrated with cells characteristic of chronic inflammation, and the epithelium will show inflammatory hyperplasia similar to that of the apical periodontal cyst Unless the relation of the cyst to a tooth is known, it is impossible to say whether a strip of cyst lining is part of a dentigerous cyst or of a periodontal cyst Cholesterol crystals are found in both cysts but they are less commonly seen in the walls of the dentigerous cyst All the other atypical features that are described under the apical periodontal cysts such as goblet cells and hyaline bodies in the epithelial lining may be found in denti-

gerous cysts The discussion on hyperplasia and neoplasia (page 66) is equally applicable to these cysts.

Primordial Cysts. Primordial cysts are believed to arise from the enamel organ either of a germ of the normal dentition or of a supernumerary tooth, before any hard dental tissues are formed Their histological appearance is the same as that of dentigerous cysts, although they

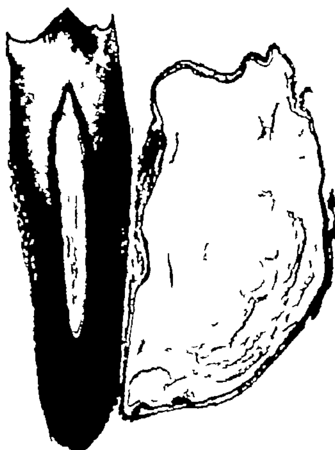


FIG. 36
Developmental periodontal cyst in association with a vital lower premolar The epithelial lining is well keratinised Haematoxylin and eosin. ×5

are not, of course directly related to any formed teeth There *is a* tendency for the cyst lining to be well keratinized (Fig 35)

Developmental Periodontal Cysts. Periodontal cysts that are developmental in origin arise from the epithelial rests of Malassez near to vital teeth, or from remnants of the dental lamina such as may be left distal to third molars. Although at operation the cysts may appear to be attached to a tooth microscopically there is healthy periodontal

membrane between cyst and tooth They may be single or multiple Figure 36 is a low power photomicrograph of one such cyst adjacent to a vital lower premolar from the mandible of a male aged 18 years There were three other similar cysts in the mandible and all teeth were vital



FIG 37

A microcyst in the gum overlying a deciduous first molar of a 8½ months foetus Haematoxylin and eosin ×24

Unless they have become infected these cysts lack any inflammatory features and the epithelial lining is well differentiated and generally shows a high degree of keratinization This mass of keratin and other degenerating cellular material imparts a cheesy character to the contents

Gingival Cysts. Many débris of the dental lamina degenerate and disappear, and others remain quite inactive, but some proliferate, become keratinized, and form cysts Keratin whorls and microcysts are commonly found in the gingivae of the human foetus and less

commonly at other ages (Fig 37) Few of these cysts reach any noticeable size but should they do so and interfere with the eruption of teeth then they are called cysts of eruption They are generally lined by an epithelial layer about two cells thick and surround a mass of keratin

Similar cysts may arise not from odontogenic epithelium but from the gingival epithelium through inflammatory hyperplasia or through trauma Ritchey and Orban (1953) described cysts formed in this manner which were traceable to the surface epithelium Epithelial rests and microcysts derived from the surface epithelium are more obviously squamous in type than those derived from the dental lamina and have well differentiated prickle-cells.

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2. BACTERIAL INVASION AND ITS SEQUELAE

Dental Caries. Dental caries is a disease affecting the hard tissues of the teeth and finally causing death of their pulps Stagnation areas, such as occlusal grooves and the interproximal surfaces around the contact points, are favoured sites for bacterial plaques and thus for the initiation of the carious process

BACTERIAL PLAQUES covering carious lesions may be large enough to be seen macroscopically or they may only be seen microscopically When stained by Gram's stain, gram positive filaments having a parallel



FIG 38
Bacterial plaque and invasion of the underlying enamel Haematoxylin and eosin ×615

arrangement may be seen against an amorphous background Gram positive coccal forms appear to be the pioneer invaders and may be seen penetrating the underlying altered enamel (Fig 38) It is important not to mistake V-shaped developmental defects containing bacteria for true invasion

THE INITIAL LESION IN THE ENAMEL Except in the presence of a lamella and artificial cracks there is no penetration of organisms prior to an alteration of the enamel structure There is initial demineralization which leaves a porous but structurally intact enamel, into which proteins and pigments from the saliva are thought to be adsorbed. These two steps must be regarded separately and may be referred to as demineralization and consolidation.

Hardwick and Manley (1952) when studying transverse serial sections after decalcification of the tooth in formic acid saturated with calcium phosphate, noted that the altered enamel showed different staining properties from normal enamel and was more resistant to decalcification (Fig 39) This latter fact often allows carious enamel to be retained

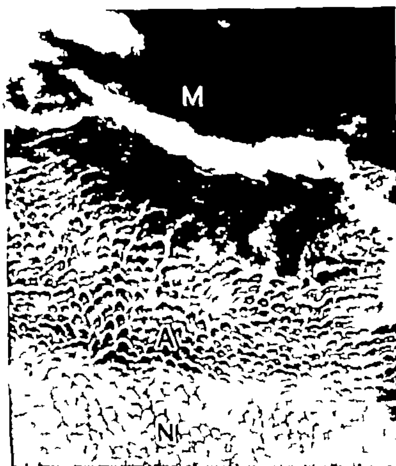


FIG. 39

Carious cavity in enamel On the surface is the closely packed area of micro-organisms and débris, M The deeply stained zone of altered enamel is shown at A, and below that is the normal enamel with well-defined prism sheaths, N Mallory's stain. ×445 (By courtesy of Dr J L. Hardwick and Professor E. B Manley *Brit dent J* 1952.)

in routine decalcified sections when all the normal enamel has disappeared.

If the process of decalcification be rapid the clinical appearance is a white chalky patch but if the process be slow and there is time for the adsorption of pigments from the saliva the lesion is represented by a brown patch

When the altered enamel is examined as a ground section, it has the appearance of being etched by an acid The enamel prisms are well

defined and the striations between the individual segments are visible The striae of Retzius appear well marked and on a smooth surface of enamel these changes tend to occur over a cone-shaped area, having its base on the surface and its point towards the amelo-dentinal junction (Fig 40) Caries beginning in a fissure will show similar changes, but due to the direction of the prisms it affects a cone-shaped area with its base towards the amelo-dentinal junction and its tip at the bottom of the fissure Since the bottom of the fissure is often near the amelo-dentinal junction, the carious process soon reaches this junction



FIG 40
Ground longitudinal section of early enamel caries seen by transmitted light The striae of Retzius and the cross striations of the prisms are well defined ×200 (By courtesy of Professor A I Darling *Brit dent J* 1956)

and then spreads laterally along it, undermining the sound enamel of the cusps In this manner the enamel may become decalcified from beneath, a process known as secondary caries of the enamel (Fig 41)

Sometimes a transparent zone in the enamel is seen below the altered enamel Mummery (1926) first described the zone and Darling (1943) has demonstrated its formation in vitro It has been thought to be a hypercalcified zone resulting from a transference of calcium salts from the altered enamel, but from studies of this zone in polarized light in air, Darling (1958) now reports that the zone is not hypercalcified at all, but at a very early stage of the carious attack

Gustafson (1957) described five zones in the established carious lesion of enamel, basing this on examination by polarizing microscope, micro-hardness tests, dark ground, transmitted and indirect illumination

(Figs 42, 43) As the carious lesion is approached from the dentine there is first a layer of normal enamel and then a layer of hypermineral ized enamel (zone 1) Next there is a zone (zone 2) in which the minerals have been dissolved out or converted to a non-crystalline state.

Nearer the surface there is another zone (zone 3) which is character ized by an increase in minerals.

In all these zones, no microscopically visible changes in the organic matrix have taken place



FIG 41

Secondary caries of enamel with caries spreading along the amelo-dentinal junction from a cavity which is to the right of the photomicrograph. The sound enamel is becoming undermined. Haematoxylin and eosin × 220

Real disintegration has begun in the next zone (zone 4) of the lesion where the minerals have been dissolved out and have disappeared, while destruction of the organic matrix has commenced

Finally (zone 5) as all the components of the enamel have been destroyed a cavity is formed

Zone 3 is an area of recalcification in which crystallites have been laid down in enamel similar to zone 2. Figure 44 the photomicrograph of an early carious lesion by transmitted light, shows Gustafson s zone 3 clearly Gustafson does not believe that the Retzius lines play any major role in the development of caries, but thinks that they only become specially prominent when disintegration of the organic matrix com mences such as in zone 4 This is contrary to the view held by Darling

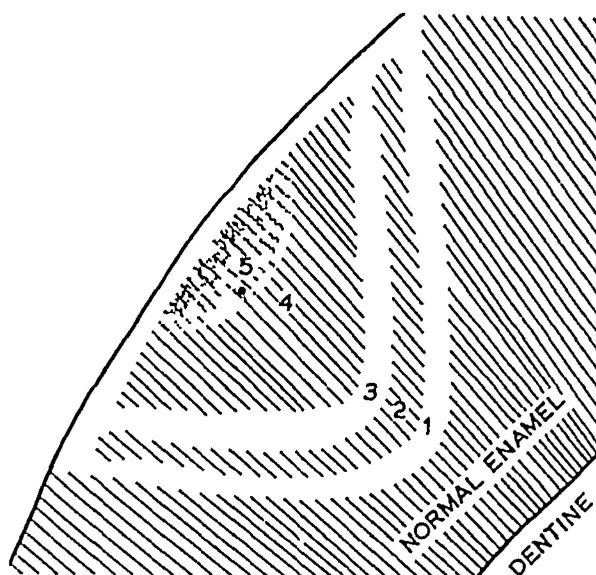


FIG 42
Schematic drawing of a carious lesion Surrounding the lesion there is a hypercalcified zone (1), which is followed by a second zone (2) containing less minerals This zone does not usually extend to the surface The third zone (3) is a zone of calcification The final disintegration of the enamel begins in zone 4 and is completed in zone 5 A harder surface layer is often found (By courtesy of Dr Gosta Gustafson *Acta odont scand* 1957)

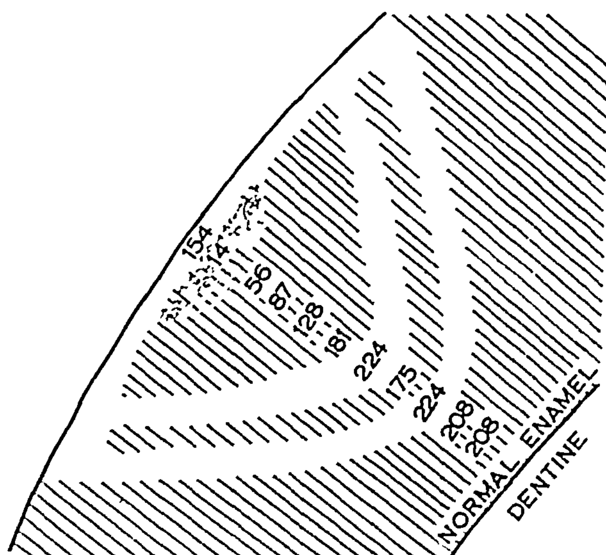


FIG 43
The results of micro-hardness tests through a carious lesion Note the increased hardness in zones 1 and 2, and also the gradual decrease in zones 4 and 5 Zone 2 shows a hardness which is lower than that in zones 1 and 3 and is even lower than the hardness in the normal enamel outside the lesion The surface layer is harder than the underlying decomposed enamel (By courtesy of Dr Gosta Gustafson *Acta odont scand* 1957)

(1958) from studies with polarized light in the various media and by microradiography who believes that the entry of caries takes place along the striae of Retzius and that the latter seem to aid the rapid spread of the lesion

Grenz ray pictures of the early lesion in which there is only altered enamel and no break on the surface show decalcification of the affected area but with a surface radio-opaque layer still present (Applebaum

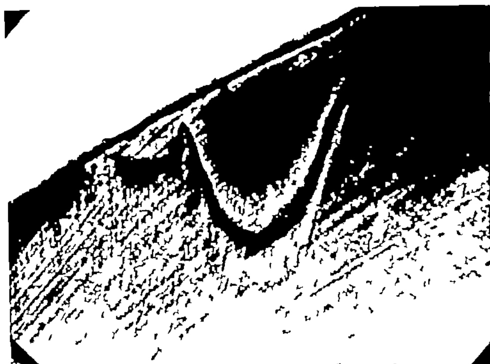


FIG. 44
Ground longitudinal section of early enamel caries seen by transmitted light. ×40

1940) It would appear that since the surface enamel is normally more highly calcified the decalcification of incipient caries still leaves more calcium salts at the surface than in the depth of the lesion (Fig 45)

Scott (1952) examined replicas of the surface of the early carious lesions and noted that sometimes there is preferential destruction of the prism sheaths and sometimes of the prisms and that it was impossible to say which was affected first.

Manley and Hardwick (1951) have shown that what were previously thought to be extensions of enamel lamellae into the dentine are in fact wedges of altered dentine under lamellae infiltrated with organisms This they regard as the result of proteolysis, but it is not always certain



FIG 45

Ground longitudinal section of early enamel caries seen by radiography It is the same section as that illustrated in Figure 40 and shows the fairly broad well calcified zone over the lesion ×200 (By courtesy of Professor A I Darling *Brit dent J* 1956)



FIG 46

Invasion of micro-organisms along prism structure in altered enamel Gram's stain ×900 (By courtesy of Dr J L Hardwick and Professor E B Manley (*Brit dent J* 1952)

that these lesions would have progressed and some may be infected cracks.

EXTENSION OF THE CARIOUS LESION IN ENAMEL. Except in lamellae, only after the enamel has been altered in the above manner can organisms be seen invading the enamel Under high magnification they may be



FIG. 47

Advanced caries of enamel Cracks have developed in the softened enamel and are filled with micro-organisms. Haematoxylin and eosin ×490.

seen penetrating along or between the prisms in decalcified sections stained by Gram's method (Fig. 46) There is the same predominance of coccal forms as in deep layers of the plaques Cracks develop in the softened enamel and become filled with organisms (Fig 47) Hodson (1952) emphasizes that care must be taken to distinguish between inter rod penetration and fine fractures between the rods for the fractures are often crescent shaped and wider at the site of commencement.

In ground sections cavitation is seen, surrounded by altered enamel

THE EARLY LESION OF CARIES OF DENTINE The pathway for invasion of the dentine by the pioneer organisms is along the dentinal tubules and their contained Tomes' processes Since there is a tendency for the



FIG 48
Micro-organisms filling the tubules The surrounding softened matrix has allowed distension of the tubules to occur, giving a beading effect Gram's stain ×490

carious process in the enamel to spread laterally along the amelo-dentinal junction, more and more tubules become vulnerable There is an initial phase of decalcification in the dentine as there is in the enamel, but there is sufficient organic matrix to retain the bulk and form of the dentine It is questionable how much any shrinkage of the affected dentine away from the amelo-dentinal junction is due to the carious process or the result of dehydration in the preparation of the section

The organisms proliferate in the tubules and generally take on three forms, either cocci, bacilli or filaments, which may be demonstrated

with Gram s stain The tubules become distended and beading occurs (Fig 48) *Pioneer organisms are often seen well in advance of the main colonies.* The affected dentine is pigmented yellow

THE VITAL REACTION TO THE EARLY LESION Even before any organ isms reach the dentine the terminal parts of the Tomes fibrils are



FIG. 49
Ground longitudinal section of two translucent zones seen by transmitted light under caries of the occlusal surface of a molar × 20

irritated and either there is calcification within the tubules resulting in a translucent zone or the fibrils die. In the latter case the affected dentine is known as a dead tract.

THE TRANSLUCENT ZONE This reaction occurs most commonly under caries of the occlusal surface of molars Fatty degeneration of the fibrils, which can only be observed in frozen sections specially stained for fat appears to precede the deposition of calcium salts Then that part of the tubules immediately under the caries becomes filled with calcium salts

acquiring a refractive index similar to the adjacent matrix and thus becoming translucent in ground sections studied by transmitted light, and dark by reflected light (Fig 49) At the same time as the odontoblasts are stimulated to organize this calcific barrier, more dentine is deposited at the pulp margin This dentine is tubular and can only be distinguished from the primary dentine by a line of demarcation If a



FIG 50
Ground longitudinal section of a button of secondary dentine corresponding to the limits of the dead tract, as seen by transmitted light
×100

dye is sealed in the pulp canal, it will penetrate the dentine up to, but not across the translucent zone

THE DEAD TRACT REACTION This is the most common reaction to caries in all sites apart from the occlusal surface of molars Fish (1932) first described this phenomenon in which irritation to the Tomes' fibrils causes death of many of the odontoblasts and in which an acellular calcific barrier which he called eburnoid seals off the pulp ends of the

associated tubules. Any odontoblasts that recover then take part in the formation of a secondary dentine which contains as many tubules as there are surviving odontoblasts. This button of secondary dentine corresponds to the limits of the dead tract (Fig. 50)



FIG. 51
Ground longitudinal section of a dead tract and button of secondary dentine under early caries of enamel, as seen by transmitted light. × 31

The tract of dentinal tubules containing the dead Tomes' fibrils usually appears black by transmitted light and light by reflected light in ground sections (Fig. 51). If a dye be sealed in the pulp canal, it is unable to pass the calcified barrier separating the secondary dentine from the primary dentine and the dead tract remains unstained.

Sometimes, when there is free lateral communication between the dead tract and adjacent tubules, the Tomes' fibrils of the latter are irritated and become calcified, so forming a translucent zone lateral and



FIG 52
Caries of dentine with transverse clefts Haematoxylin and eosin ×210



FIG 53
Caries of dentine with liquefaction foci and the tubules in the adjacent softened dentine bent round them Haematoxylin and eosin ×120

parallel to the dead tract In this manner the dead tract is sealed off completely not only from the pulp but from adjacent vital dentine

TRANSITION FROM THE TRANSLUCENT ZONE TO THE DEAD TRACT When the translucent zone forms other than under occlusal caries of a molar such as under cervical caries it is generally limited in extent and further stimulation to the odontoblasts results in their death the

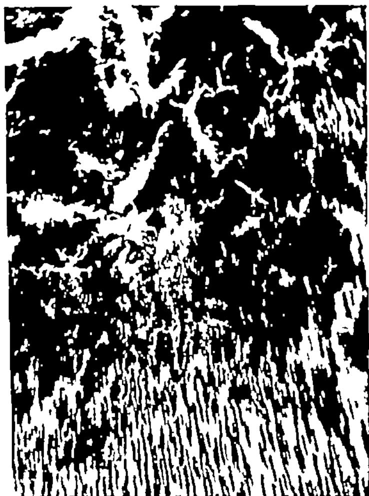


FIG 54
Advanced caries of dentine with cavity formation in the softened dentine Haematoxylin and eosin × 120

deposition of eburnoid and secondary dentine In this manner a dead tract forms under the translucent zone

ADVANCED CARIES OF DENTINE. As the tubules of the carious dentine become more distended they fuse and spaces known as liquefaction foci form in the softened matrix Both transverse and vertical spaces form, the former often along the incremental lines and less commonly the lateral processes of the tubules (Fig 52) The tubules in the adjacent

softened dentine are bent round the foci (Fig 53) Finally, with destruction of the softened dentine, the latter crumbles and a large cavity is left (Fig 54)

When the carious process reaches the secondary dentine, the organisms proliferate in any tubules that may be found crossing the



FIG 55
Pioneer organisms proliferating in the tubules crossing the barrier between the primary and secondary dentine Haematoxylin and eosin ×65

barrier between it and the primary dentine (Fig 55), and laterally along this barrier, splitting the two layers (Fig 56) When the tubules are few and very irregular, the attack is slow and mainly frontal and layer by layer

CENTRAL CARIES After the organisms have reached the pulp, adjoining dentine, often secondary dentine, becomes invaded from the pulpal aspect and the caries progresses peripherally The fibrillar structure of the secondary dentine is remarkably well demonstrated in these

sections. Lateral clefts form along incremental lines from the carious pulpal walls across the tubules. This type of caries is found in roots with open pulp canals where crowns have broken off after having been weakened by caries.

CARIES OF CEMENTUM In cervical caries following exposure from age or periodontal disease organisms thriving in the organic material of the pocket attack the cementum by a combination of proteolysis and decalcification (Fig. 57) The Sharpey fibre bundles form pathways between the incremental lines and the latter split and become under



FIG 56
Organisms proliferating laterally along the barrier between the primary and secondary dentine, splitting the two layers. Haematoxylin and eosin. ×120

mined Hodson (1952) believes that the bacteria enter fine splinter lines parallel with the cemental fibres In this manner the cementum is gradually destroyed and the underlying dentine shows either a dead tract or a small translucent zone with transition to a dead tract.

ARRESTED CARIES Should the walls of a carious occlusal cavity collapse and the softened dentine be removed by attrition, the affected surface becomes smooth and polished The dentine in this area will show a well marked translucent zone

Inflammation of the Pulp. Due to its distinctive anatomical position within the tooth, the chances of the pulp overcoming a bacterial infection

are few One of the essential features of any inflammatory process oedema, and oedema in a confined space will compress capillaries a veins thus reducing any chance of repair While hyperaemia may reversible, acute inflammation usually ends in necrosis of the pulp



FIG 57
Caries of cementum Haematoxylin and eosin
×220

It is convenient to consider inflammation of the pulp under three main headings subacute, acute and chronic inflammation The organisms concerned in dental caries and their toxins are the commonest causes of inflammation of the pulp, but there is strong clinical evidence that just occasionally a pulp may become infected by bacteriaemia Fracture of a tooth, and accidental exposure during cavity preparation may also allow organisms access to the pulp Minute cracks in the crown of a tooth due to trauma must be borne in mind when there is no obvious cause for pulpitis

Injury to the dentine during cavity preparation chemical irritation from cements and thermal stimuli through unlined metallic fillings may also cause inflammation in the pulp and these will be considered



FIG. 58
Subacute pulpitis. A small focus of lymphocytes and dilated capillaries are separated from the dentine at the top of the picture by normal tissue Haematoxylin and eosin ×95

separately In advanced periodontal disease the pulp may become infected through the apical canal

SUBACUTE PULPITIS Subacute inflammation of the pulp may precede acute inflammation, or under favourable conditions it may result from a partial resolution of acute inflammation The latter change will be considered after acute pulpitis

There is little doubt that when the toxins of caries producing organisms stimulate the pulp to lay down secondary dentine under a dead tract or a translucent zone in the dentine, there is a mild inflammatory reaction with capillary dilatation and a mild lymphocytic

infiltration This may quickly resolve or may remain *in statu quo* indefinitely, or may with the advance of caries progress to acute inflammation Small foci of lymphocytes may be found in the pulp sometimes separated from the dentine by normal tissue (Fig 58) In a cornu of the pulp under a carious cavity or under an exposure that



FIG 59
Subacute pulpitis A focus of chronic inflammatory cells localised to one cornu of the pulp chamber in relation to caries involving the secondary dentine Haematoxylin and eosin × 110

has been capped, an abscess may form (Fig 59) This becomes localized and is walled off by a condensation of fibrous tissue, while the rest of the pulp remains normal As Herbert (1945) has shown, such a localized chronic abscess may cause pain, yet the tooth give a normal response to vitality tests He found a number of teeth that were symptomless yet on section showed a chronic abscess

In many cases the chronic inflammatory reaction eventually extends throughout the whole pulp, which is replaced by granulation tissue

infiltrated with plasma cells and lymphocytes (Fig 60) There may be multiple chronic abscess cavities in such a pulp The odontoblasts being so specialized are lost early but their pyknotic nuclei may remain

ACUTE PULPITIS Should the caries progress, the pioneer organisms reach the pulp and an acute inflammation supersedes the subacute



FIG 60
Subacute pulpitis. Almost the whole pulp is infiltrated with plasma cells and lymphocytes Haematoxylin and eosin. × 130

Hyperaemia of the pulp (Fig. 61) is soon followed by an exudate of fluid and cells (Fig. 62) Polymorphonuclear leucocytes are predominant and are most evident around acute abscess cavities containing necrotic pulp tissue, dead leucocytes and organisms In Gram stained sections organisms will be seen wherever the inflammation is most acute Cultures will reveal a predominance of *Streptococcus viridans* and *Staphylococcus albus* and *aureus* If there is an exposure allowing drainage the acute pulpitis may be restricted to the coronal part of



FIG 61

Hyperaemia of the pulp The dentine is to the left in the photomicrograph, the dilated capillaries to the right Haematoxylin and eosin × 110

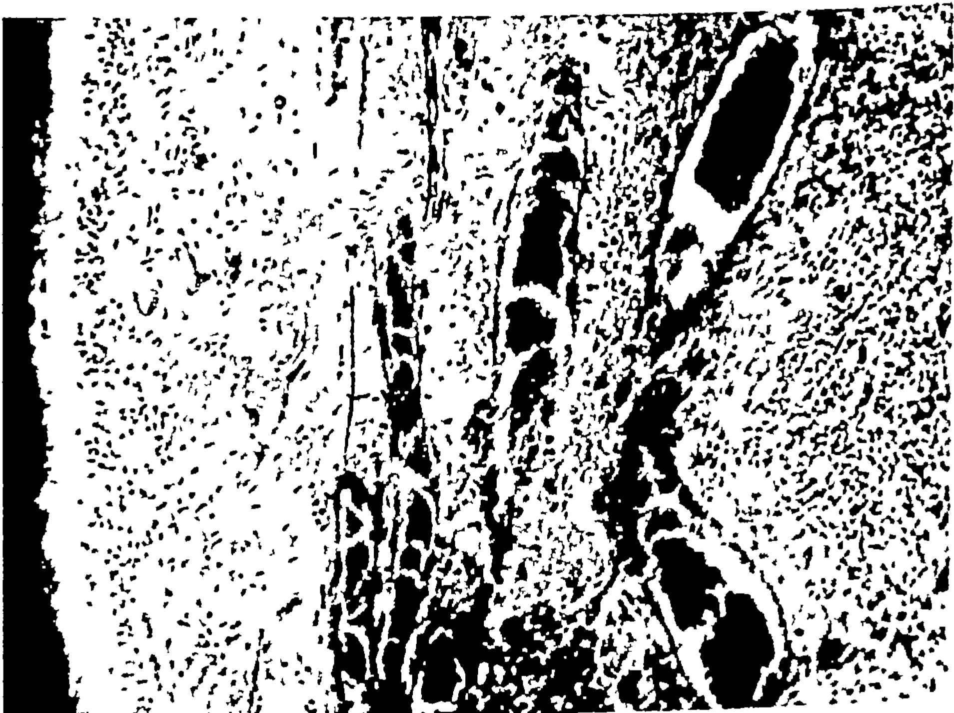


FIG 62

Early ac[illegible] pulpitis The dentine is to the left in the photomicrograph the capi[illegible] dilatation and polymorphonuclear leucocytic infiltration to the right Haematoxylin and eosin × 110



FIG. 63

Acute pulpitis with abscess cavity. The abscess cavity appears empty because the pus has drained through an exposure not shown in the photomicrograph. Haematoxylin and eosin. ×34



FIG. 64

Acute pulpitis of the pulp chamber under a carious cavity (top left) which is penetrating the secondary dentine. Haematoxylin and eosin. ×34

the pulp (Fig 63), but as a general rule the inflammation spreads until the whole pulp is involved, and necrosis quickly follows (Fig 64) The dead tissue loses its staining properties and stands out in contrast to the haematoxyphil nuclei of the still vital densely packed leucocytes



FIG 65
Necrosis of pulp The densely packed degenerating polymorphonuclear leucocytes near the apex at the bottom of the photomicrograph are in contrast to the pale necrotic pulp above Haematoxylin and eosin × 110

(Fig 65) According to Bulleid and Shuttleworth (1949) culture necrotic pulps show besides the caries-producing organisms, s cocci staphylococci, *Micrococcus tetragenus*, sarcinae, yeasts and of the mesentericus or proteus groups

Under certain conditions, namely a wide exposure with good resistance and organisms of low virulence, a balance may be resulting in the following forms of chronic pulpitis

CHRONIC ULCERATIVE PULPITIS Should there be drainage th a wide exposure before necrosis follows an acute pulpitis, the

inflammatory reaction may be localized to the region of the exposure (Fig. 66) The apical portion of the pulp will consist of granulation tissue infiltrated with lymphocytes plasma cells and histiocytes These pulps often have many calcific deposits following degenerative changes in the apical pulp cells

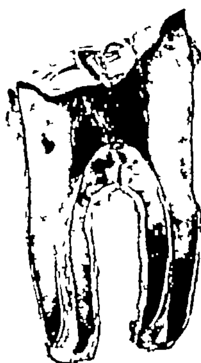


FIG 66

Chronic ulcerative pulpitis This deciduous molar has a wide exposure and the pulp chamber is full of granulation tissue densely infiltrated with chronic inflammatory cells. Haematoxylin and eosin. ×6.



FIG. 67

Chronic hyperplastic pulpitis The granulation tissue fills the carious cavity and is covered with epithelium. Haematoxylin and eosin ×4 5

CHRONIC HYPERPLASTIC PULPITIS (PULP POLYP) Under conditions such as may be found in deciduous molars with wide apices and large exposures, the pulp may respond to the infection at the exposure by a proliferation of granulation tissue that may fill the carious cavity At first the surface of the granulomatous polyp will be ulcerated with necrotic tissue resting on granulation tissue infiltrated with polymorphonuclear leucocytes In the older lesion the inflammatory reaction on the surface subsides and the latter becomes epithelialized by direct contact with adjoining gingival epithelium or by grafts of epithelial cells implanted on its surface (Fig. 67) The body of the polyp consists

of fibroblasts and vessels arranged at right angles to the surface containing focal collections of lymphocytes and plasma cells There is often much secondary dentine formation and pulp stones form in the pulp cavity beneath the polyp Epithelium from the surface of the polyp may line the carious cavity and so provide a seal to the pulp canal preventing infection of the pulp by organisms from the oral cavity The apical portion of the pulp may be normal or it may be replaced by granulation tissue.

EFFECTS OF CAVITY PREPARATION AND FILLING MATERIALS The reaction of the pulp to the mechanical trauma of cavity preparation and to the irritant effect of various filling materials differs from that to caries as follows (Manley, 1950)

'Under caries and attrition the tubules are opened gradually from the surface, stimulation occurs, and the pulp has time to lay down secondary dentine which seals off the affected tract of tubules from the pulp This is a *constructive* reaction In operation procedures an entirely different set of circumstances prevails Preparation of the cavity involves the opening up suddenly of a large number of tubules at considerable depth and extending beyond the carious limits As acid cement comes into contact with these newly opened healthy tubules which are in free communication with the pulp, the irritant is able to reach the pulp without hindrance, and a *destructive* reaction results'

Fish (1932) noted that in carefully prepared experimental cavities, the dead tract reaction occurred in the tubules cut across, and secondary dentine formed as a specific reaction to the injured fibrils Adjacent uninjured tubules were never affected The severity of the pulp reaction varied with the degree of injury to the dentinal fibrils, and if the cavity was injudiciously prepared an acute pulpitis resulted

Zander (1946) demonstrated how filling materials that produce a reaction in the pulp when placed in cavities prepared in sound teeth, do not do so when inserted in cavities where caries or attrition have produced secondary dentine Many of the earlier investigations were carried out on sound teeth in young people that had to be extracted for orthodontic reasons In these there was no secondary dentine and the dentine was very permeable In fact the younger the tooth and the deeper the cavity, the more severe was the reaction Manley (1936) used the teeth of dogs and in 1941 human teeth, employing a careful cavity preparation technique to minimize the traumatic effect and described the reaction of the pulp to different filling materials Zinc oxide and eugenol produced no reaction, while silicate cements produced an acute inflammatory reaction within 24 hours According to the degree of irritation, recovery takes place and secondary dentine

is laid down, or an abscess forms and the whole pulp may become necrotic. The reaction under the zinc phosphate cements was less severe than under copper phosphate cements or silicates. It appears that the surface acidity of the cement at the time of insertion in the cavity is the critical factor. A constant finding with all observers was that pain was not related to the degree of damage to the pulp and that quite severe reactions were often symptomless. There is no doubt that many of the mild reactions of the pulp are followed by recovery.

More recently Kramer and McLean (1952) have studied the effects on the pulp of unlined self polymerizing acrylic restorations and they tried to assess the pulp reactions to the various resins by measuring the polymorphonuclear infiltration in the odontoblast layer, the degree of focal or diffuse chronic inflammatory infiltration and the distribution of the fluid content. Occasionally large blisters formed in the pulp directly under the affected tubules, and odontoblasts were aspirated into the tubules.

AERODONTALGIA. During high altitude flying, teeth that are symptomless at sea level may on occasion become painful. Such teeth always have some pathological condition in their pulps as a result of caries or recent cavity preparations. The cause of this pain during decompression is thought to be the release of nitrogen bubbles from the blood and tissue fluid into a pulp with impaired circulation. Because of the impaired circulation the pressure is not equalised quickly enough to prevent pain. Orban and Ritchey (1945), in an extensive clinical and histological study of teeth that became painful on decompression, found that of 75 teeth 16 had oedematous pulps, 17 had acute pulpitis, 15 had chronic pulpitis and 7 had non vital pulps. In the remaining 20 cases, 3 were teeth with normal pulps and in the remainder the pathological condition was difficult to classify. Teeth with normal pulps did not hurt under decompression whether the tooth was intact, carious or filled. Teeth with open cavities did not cause pain on decompression even if the pulp was diseased, suggesting that pressure was equalized through the dentinal tubules. Harvey (1943) found that many teeth painful on decompression had recently been filled, and he thought this was due to the mild hyperaemia following heat production while burring. While a zinc oxide and eugenol sedative dressing as a lining to a cavity following preparation will prevent pain during decompression, such a measure will not of course cure pathological conditions of the pulp such as Orban found in his series of cases.

Apical Granuloma. This is a mass of granulation tissue, inflammatory cells and epithelial cords that has replaced the apical part of the periodontal membrane and is found in association with a dead or dying

pulp. Almost every cell type associated with inflammation and repair may be found in this lesion and the picture will vary considerably according to the stage reached by the lesion when it is removed and examined microscopically

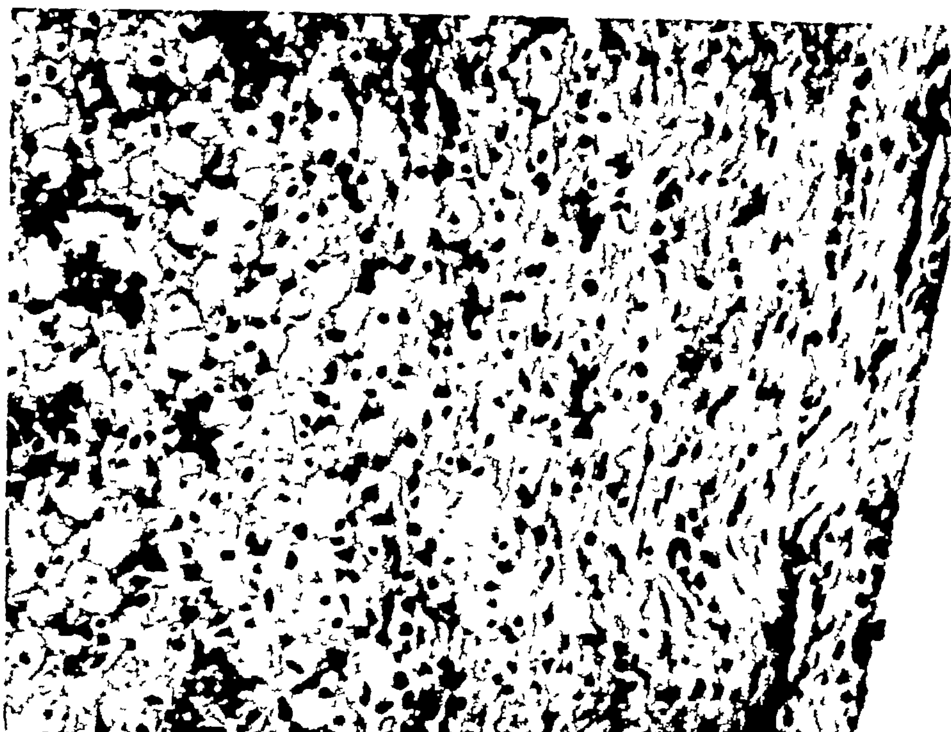


FIG 68
Apical granuloma with foam cells at the left side of the photomicrograph
Haematoxylin and eosin ×200

THE YOUNG APICAL GRANULOMA—ACUTE INFLAMMATORY STAGE Even while the pulp is still vital but acutely inflamed, the apical portion of the periodontal membrane will be oedematous and lightly infiltrated with lymphocytes and plasma cells Then as the organisms and their toxins from the pulp canal reach the a ical area, small areas of necrosis occur, around which there is a d [illegible] ation of neutro [illegible] oly-morphonuclear leucocytes Small [illegible] of necrotic [illegible] removed by the histiocytes, but sh [illegible] tion be very necrotic tissue will liquefy and, to [illegible] he dead or polymorphs, will form pus and dr [illegible] ugh the r through a sinus to the surface.

THE [illegible] ILD APICAL GRA [illegible] REPAR [illegible] While [illegible] omata form from [illegible] of a s abscess, [illegible] these often sh [illegible] fo 68) Th [illegible] a histiocyte ac [illegible] with deg [illegible] oducts.

New capillaries and fibroblasts proliferate from the margins of the lesion and while the polymorphonuclear infiltration diminishes, the lymphocytic and plasma cell infiltration increases At the same time there is an inflammatory hyperplasia of the epithelial rests of Malassez. These rests proliferate in cords and trabeculae throughout the granulation tissue and often along the margin of the root which may show evidence



Fig 69
Apical granuloma with polymorphonuclear leucocytes almost obliterating the outline of the epithelial trabeculae Haematoxylin and eosin. ×75

of osteoclastic resorption The granulation tissue and the epithelial strands may even proliferate through the apex of the tooth into the pulp canal.

Most of the polymorphs at this stage are attracted to the proliferating epithelium where they are seen in varying stages of degeneration in the intercellular spaces between the prickle cells sometimes in such numbers as almost to obliterate the outline of the latter (Fig 69) The epithelial strands often encircle dilated capillaries or foci of plasma cells. As when there are collections of plasma cells elsewhere in the body mulberry shaped brightly eosinophilic bodies known as Russell bodies are frequently seen and they are thought to be derived from the degenerating nuclei of the plasma cells

Foreign body giant cells may be present singly or grouped around clefts from which cholesterol crystals have been removed in the preparation of the paraffin section

At the periphery, fibroblasts and collagen fibres are arranged around the granuloma in a concentric fashion and adjacent bone undergoes resorption

THE OLD APICAL GRANULOMA—LATE REPARATIVE STAGE. This is not often seen unless the focus of infection, namely the dead tooth, has been extracted or the root canal filled, leaving the granuloma behind Then the fibroblasts lay down dense collagen bundles, the inflammatory hyperplasia of the epithelium subsides and, except for an occasional focus of lymphocytes, it is just a fibrous scar.

Periodontal Cysts (*Dental Cysts*) are cysts lined with epithelium derived from the rests of Malassez These epithelial rests are the residues of specialized odontogenic epithelium and this accounts for some of the distinctive characters of the cysts They are the commonest cysts in the jaws associated with the permanent dentition The majority are infective in origin and form at the apices and less commonly lateral accessory canals of dead teeth They result from inflammatory hyperplasia of the epithelial rests in apical granulomata, although why a cyst forms in some granulomata and not in others is not understood There would not appear to be any evidence that lack of blood supply accounts for the death of the central cells in a mass of epithelium

Less commonly periodontal cysts occur that are quite unrelated to apical infection and are developmental in origin (see page 29)

APICAL PERIODONTAL CYSTS The young periodontal cyst is a cavity lined by hyperplastic stratified squamous epithelium in an apical granuloma which still shows subacute inflammatory features The epithelium is irregular in thickness and many strands proliferate into the supporting fibrous tissue There is both intercellular and intracellular oedema, and many polymorphonuclear leucocytes fill the spaces between the prickle cells The superficial degenerating prickle cells and the polymorphonuclear leucocytes are shed into the cyst cavity There are many dilated capillaries adjacent to the basal layer of the hyperplastic epithelium giving to the epithelium a lattice-like appearance as it encircles them as epithelial cuffs The cyst cavity also contains foam cells that have migrated through the epithelium (Fig 70) These are sometimes found in large numbers in the surrounding fibrous tissues, together with focal collections of plasma cells and lymphocytes The fibrous tissue is arranged concentrically around the cyst, but it may contain trabeculae of epithelium some of which form small cysts that later fuse with the larger one

As the inflammation subsides, the epithelium becomes even in thickness sometimes with a well-differentiated basal and prickle cell layer, but at other times more undifferentiated and embryonic looking (Fig. 71) Occasionally there is a well-marked keratin layer

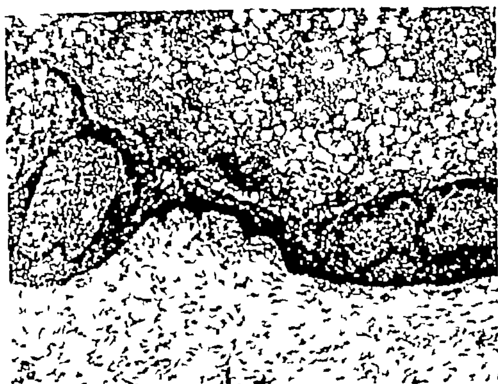


FIG. 70
Apical periodontal cyst with inflammatory hyperplasia of the stratified squamous epithelial lining and its cavity full of foam cells and polymorphonuclear leucocytes. Haematoxylin and eosin × 105

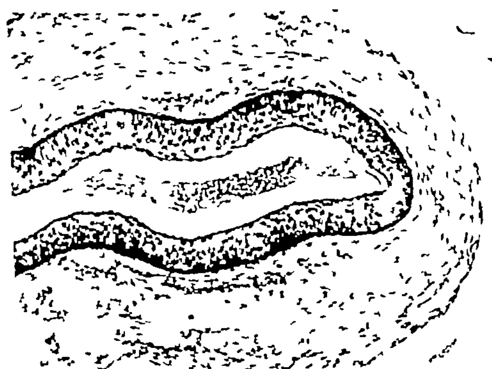


FIG. 71
Apical periodontal cyst with a lining of well-differentiated stratified squamous epithelium supported by fibrous tissue lacking inflammatory features. Haematoxylin and eosin × 125

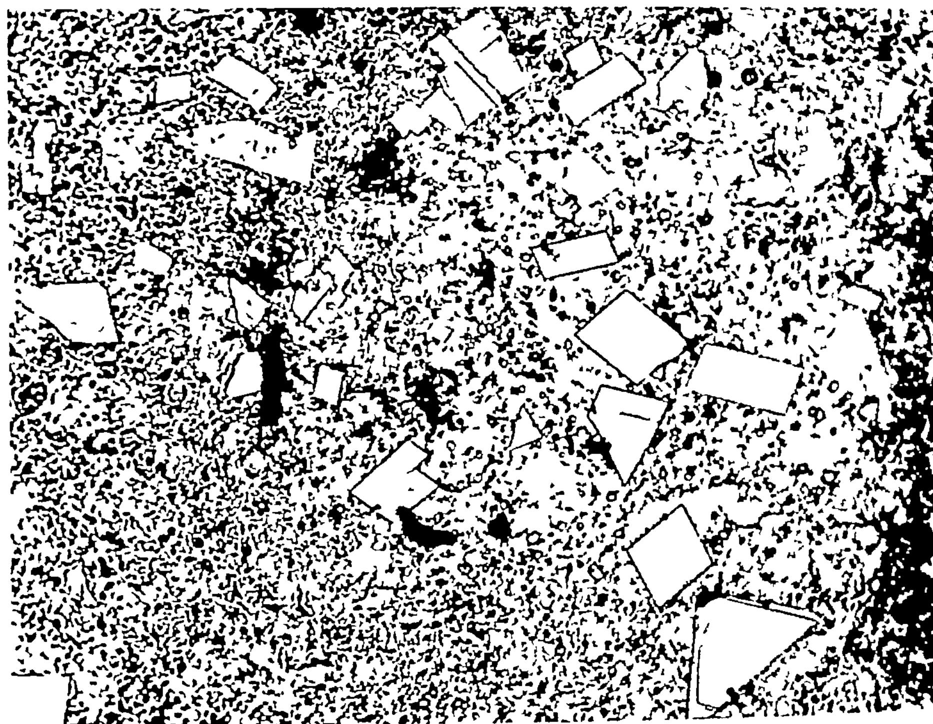


FIG 72

Contents of apical periodontal cyst Cholesterol crystals and epithelial débris ×110

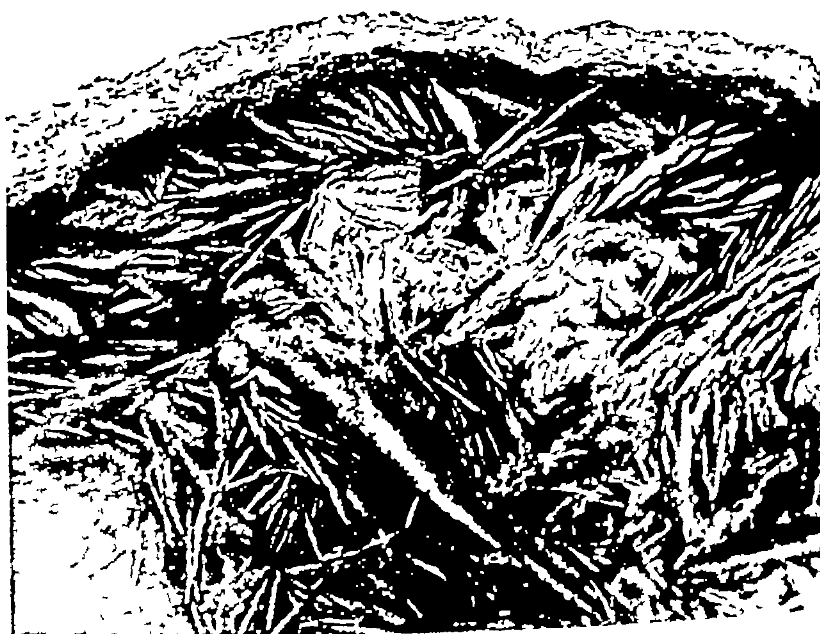


FIG 73

Clefts in the cavity of an apical periodontal cyst, from which the cholesterol crystals have been dissolved in the preparation of the paraffin section Haematoxylin and eosin ×75

Cholesterol crystals are numerous in these cysts and they give a metallic sheen to the contents They are rhomboidal in shape and are readily identified in a fresh smear (Fig 72) In paraffin sections clefts left by the cholesterol crystals are seen both in the fibrous tissue wall and in the cyst cavity (Fig 73) Those in the cyst wall are often surrounded by foreign body giant cells and they may be so numerous as to give a cribriform pattern to the wall (Fig 74)

Neighbouring trabeculae of bone undergo osteoclastic resorption on the side towards the cyst while there is bone apposition further away



FIG. 74

Foreign body giant cells adjacent to cholesterol clefts in the wall of an apical periodontal cyst Haematoxylin and eosin × 125

The apposition does not keep pace with the resorption and despite new sub-periosteal bone formation it is not enough to prevent the final perforation of the bony plate by the cyst.

MULTIPLE PERIODONTAL CYSTS Occasionally a number of these cysts may be found in the jaws of the same patient. These may arise from multiple granulomata or as daughter cysts by epithelial proliferation in the wall of a cyst. When a cyst is marsupialized, the remaining epithelium assumes mature characteristics and becomes keratinized Knight and Manley (1955) in discussing the formation of multiple dental cysts, feel that since little is yet known of the reactions of these residues of

NEOPLASIA OR HYPERPLASIA IN PERIODONTAL CYSTS While it is a theoretical possibility that an adamantinoma may arise from neoplasia supervening in the epithelial lining of a periodontal cyst, since both lesions may arise from the same odontogenic epithelium, such cases must be very rare. Lucas (1954) could not find any such example in a study of 193 periodontal cysts, 41 dentigerous cysts and 28 adamantinomata Great care must be taken to distinguish epithelial hyperplasia from neoplasia It is not uncommon to see irregular epithelial proliferations, either as isolated epithelial follicles in the connective tissue of the cyst wall (Fig 78), or as strands of epithelium continuous with the cyst lining (Fig 79) Careful study of the proliferations will show that although superficially similar to the adamantinoma, they do not in fact show the same cell differentiation In doubtful cases, serial sections of the whole cyst must be studied since the characteristic structure of an adamantinoma may not be evident in one of its larger cysts

THE INCREASE IN SIZE OF CYSTS OF THE JAWS Toller (1948), in a series of carefully conducted experiments measuring the intra-cystic fluid pressure in uninfected cysts, found that the average pressure in 51 periodontal cysts was 70 cm of water and in 9 dentigerous cysts was 65 cm of water Using an asometer, he showed that a freshly dissected cyst wall behaved as a membrane impermeable to colloids (albumen) and permeable to crystalloids and water The experiments indicated that the cause and maintenance of the positive pressure in the cyst was related to the osmotic tension of the cyst fluid Toller believed that the result of the breakdown of complex tissue proteins into large numbers of molecules of more simple proteins would account for the continued supply of substances to maintain hypertonicity as the cyst grows larger

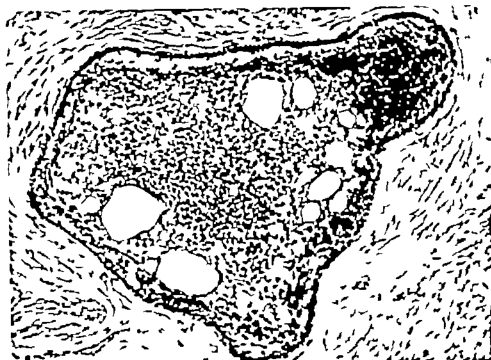


Fig. 78
An island of hyperplastic but not neoplastic epithelium in the wall of an apical periodontal cyst, superficially resembling an adamantinoma. Haematoxylin and eosin. × 120

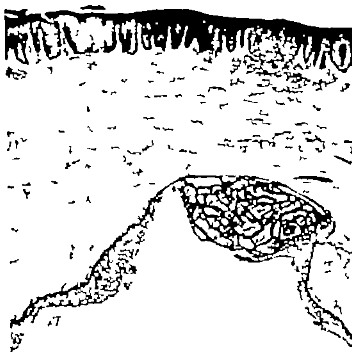


Fig. 79
Irregular epithelial proliferations in the wall of an apical periodontal cyst. Haematoxylin and eosin × 24

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3 REPAIR AND REGENERATION

TRAUMA TO DEVELOPING TEETH

Incompletely formed teeth which have not yet erupted are frequently affected by mechanical injuries

The commonest effect is that the part of the tooth already formed at the time of injury is dislocated without serious damage to its vascular supply The root then continues to form in the original direction so that

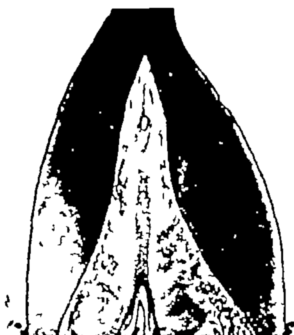


FIG 80

Upper central incisor injured by a fall at 6 years of age A very marked incremental line corresponds to the date of injury Picrothionin. ×8

on completion the tooth presents an abruptly angulated form These dilacerated teeth show no particular histological abnormalities except alteration in alignment of tissues.

If however the injury be such that the vascular supply of the growing tooth is damaged, the temporary interruption is reflected in qualitative changes in the dental hard tissues. In the completed dentine this is seen as a very well marked incremental line (Fig. 80) at which all or most of the original dentinal tubules stop and at which the original predentine layer can often still be recognised (Fig 81) After an interval of time the blood supply is evidently restored by formation of new vessels and apposition of dentine is usually resumed While at

first this may consist of matrix without tubules and containing cellular elements, it later contains the branched ends of new dentinal tubules and subsequently well-formed dentine (Fig 82) The coronal pulp cavity becomes very small or obliterated The junction of the old and new



Fig 81
Same specimen as Figure 80 Enlarged view showing original predentine still recognisable The tubules of the old dentine on the left terminate at this point New dentine with new tubules has been subsequently deposited Picrothionin ×65

tissues is sometimes weak so that teeth may afterwards fracture at this point, the crown portion exhibiting a conical depression and the root a conical prominence

In cases where a great part of the tooth is already compl the time of injury, the formation of good tubular dentine may be resumed in the crown, but the coronal pulp may become by a calcified tissue resembling primitive bone

It is to be supposed that the period during which the blood is interrupted is such that odontoblasts are unable to survive, other pulp cells of less exacting requirements are able to do so u blood supply is restored In the less mature parts of the too

odontoblasts may later be differentiated or in those parts nearer the apex the old odontoblasts may recover

In very early injuries to the tooth germ the shape of the crown and the arrangement of its tissues may be altered Thus may be caused



FIG. 82
Decalcified tooth stained with picrothionin Normal dentinal tubules (black) are seen in the upper part of the figure· below this is a line of matrix without tubules (white) then the terminal branches of new tubules extending towards the pulp. × 120

hypoplastic defects of the enamel or jagged protrusions of dentine where the dentinal papillary substance has herniated as the result of pressure (odontocele) Occasionally this may result in the formation of an additional, well-co-ordinated enamel-covered cusp or denticle joined to the same root (Fig. 83) It has been shown experimentally in animals that the very early tooth germ has remarkable powers of recovering from mechanical injury so that even half a germ can develop into a whole tooth of normal pattern (Glasstone 1952) In man however most injuries appear to occur at a stage after this regulative power is lost.



FIG 83
Injury to germ of lower central incisor at 2 years In addition to the usual line in the dentine corresponding to the time when the tip of the crown was dislocated labially an extra crown has formed on the labial side (*Brit. dent J* 1958) Picrothionin ×5

TRAUMA TO FULLY FORMED TEETH *

Without Fracture of the Tooth. HAEMATOMA OF THE PULP Haemorrhage into the pulp may form a haematoma beneath the predentine with disruption of the odontoblast layer and many red cells may be forced up the dentinal tubules (Figs 84, 85) It is presumed that such a haematoma is eventually absorbed or calcified Small haemorrhages into the pulp are frequently seen in extracted teeth and are caused during the operation of removal.

ASEPTIC NECROSIS If the apical vessels are torn and the blood supply to the pulp does not become re-established, the pulp undergoes aseptic necrosis The pulp loses its staining properties and exhibits in sections a 'ghost' of its normal structure. After a variable period of time the necrotic tissue usually becomes infected by organisms derived either from the blood stream or from neighbouring sites

* The effects of small repeated injuries producing attrition or abrasion are considered under regressive changes (page 94)



FIG 84
Haematoma of pulp from the first lower permanent molar of a child. (*Brit dent J* 1949) Haematoxylin and eosin ×22.

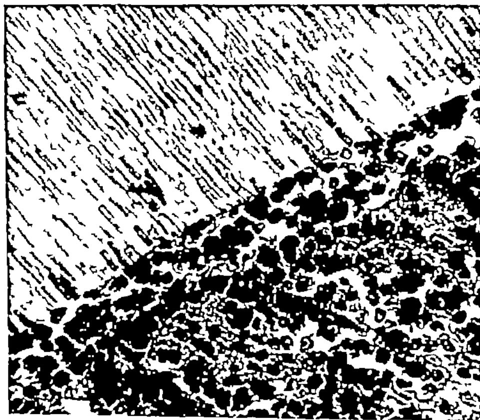


FIG 85
Detail from Figure 84 ×400 showing erythrocytes forced up the tubules of the predentine (*Brit dent J* 1949)

CALCIFICATION OF THE PULP AND INTERNAL RESORPTION OF DENTINE If the apical vessels be injured but the blood supply subsequently becomes restored, the tooth may remain vital but show various structural changes In the crown the pulp may never recover and remain atrophic,



FIG 86



FIG 87

FIG 86—Trauma to fully formed teeth Upper central incisor from a girl of 15 There is an oval area in the root where dentine has been resorbed and almost completely repaired by new dentine, though the pulp and periodontal membrane communicate by the opening seen on the left Picrothionin, ×5 (*Brit dent J* 1956)

FIG 87—Cervical region of tooth seen in Figure 86 showing older dentine on the left, former predentine layer, and covering of almost homogeneous calcified matrix There are bacterial growths in the pulp cavity which became infected Haematoxylin and eosin ×110 (*Brit dent J* 1956)

perhaps with some apposition of calcified matrix In the root the restoration of the blood supply may be accompanied by resorption of dentine from the pulp side which may even perforate the side of the tooth This stage is followed by one of repair, in which the resorbed dentine is replaced by calcified matrix resembling either primitive bone or cementum, or in the case of young teeth sometimes tubular dentine

(Figs 86 87 88) Teeth which survive long enough may become practically solid throughout and insensitive (Figs 89 90)

When resorbed dentine is repaired with tubular dentine the earliest formed repair tissue usually contains no tubules but occasionally it may do so (Fig. 91) so that the new tubules start from within the resorption bays of the old dentine It is not known whether the odontoblasts concerned are the old odontoblasts which have recovered, or new odontoblasts differentiated from available connective tissue cells, or both

With Fracture. If the line of fracture is such that the pulp is exposed to the oral fluids the result in the absence of treatment will be infective pulpitis and ultimately gangrene of the pulp (Fig. 92)

If the line of fracture is such that the pulp is only separated from the oral fluids by a very thin layer of dentine the result in the absence of treatment will be the same since the thin dentine will be permeable

When the line of fracture is through the crown but separated by a sufficient thickness of dentine from the pulp the pulp will react by the apposition of secondary dentine and after a period of mild inflammation the pulp will return to normal

When the line of fracture is through the root and does not communicate with the oral fluids, there exists the possibility of union provided that mobility is restricted and infection is not present In many cases, however repair is limited to the formation of a false joint (Fig 93) The sequence of events is analogous to that occurring in fractures of bones

At the time of injury there is haemorrhage between and around the two or more fragments. The pulp is damaged but the nerve bundles at least will probably not be torn through The haematoma becomes organized The edges of the fragments become resorbed by osteoclasts and on the resorbed surfaces cementum is deposited (Fig. 94) If mobility is limited for example by splinting the apposition of cementum may ultimately unite the fragments More commonly cementum covers the fractured surfaces in such a way that they fit together but remain separated by a layer of fibrous tissue, making a false joint (Fig 93) though some of the smaller fragments may become firmly united (Fig 95) The open ends of the pulp chamber nearest the fracture become occluded by dentine and cementum except for an opening through which the nerve bundle and vessels pass between the root and coronal fragments The condition of the pulp in the root fragment usually remains good but that of the coronal fragment shows various degrees of fibrous degeneration or atrophy In these repair reactions the blood supply is derived principally from the periodontal membrane and to some extent from the vessels of the pulp in the root fragment.

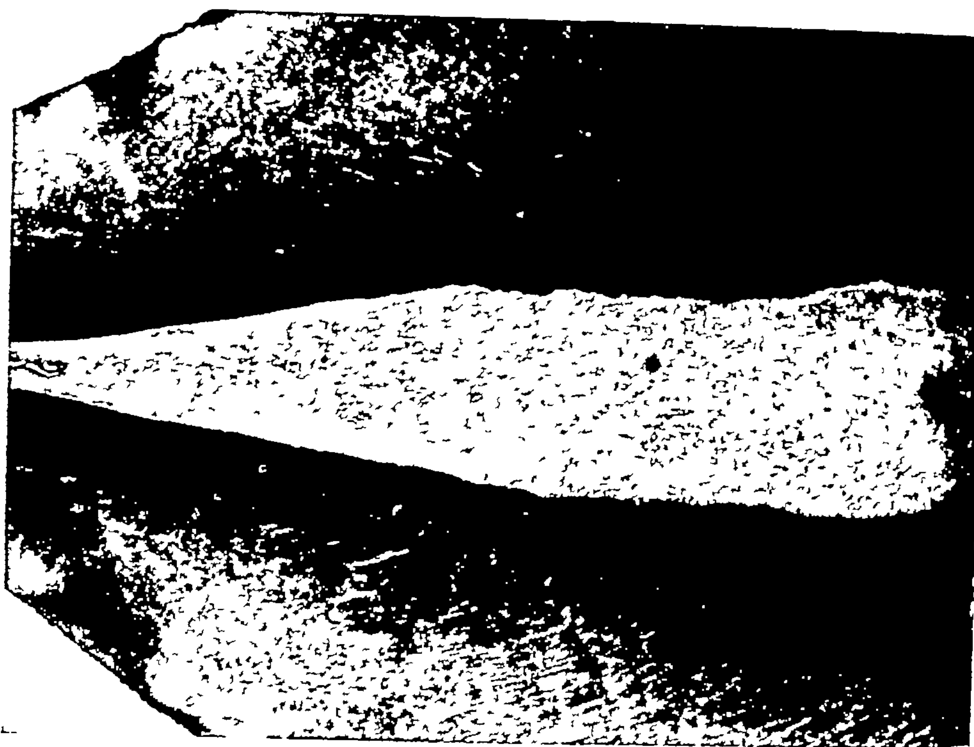








FIG. 91
Resorption of dentine has been repaired at once with new tubular dentine Picrothionin × 150 (*Proc roy Soc Med* 1952.)



FIG. 92
Fracture with exposure of pulp A premolar has been split vertically by some accident and infection has reached the pulp in which an abscess has developed. As there was no visible separation of the frag ments, the fracture had not been recognised clinically Haematoxylin and eosin × 10



Fig 93
Fracture through the root of a tooth fibrous union The fractured surfaces have been partly resorbed and resurfaced with cementum The interval between the two parts of the tooth is filled with fibrous tissue containing also some bone trabeculae Haematoxylin and eosin ×5



Fig 94
Tooth fractured through root The surface of dentine exposed by fracture has been resorbed and a layer of cementum has subsequently been deposited The two hard tissues are separated by a line which stains deeply with haematoxylin Haematoxylin and eosin ×100



FIG. 95
Fractured root Several small fragments have become united by cementum which has filled the cracks and covered the surfaces. Picrothionin. × 50



FIG. 96
Fracture of cementum. As the result of a sudden strain a small piece of cementum has been torn from the sloping surface of the root R by the periodontal fibres attaching it to the alveolar crest B Silver impregnation × 140

Fracture of Cementum As the result of a sudden strain, such as biting on a lead shot, a portion of cementum may become torn from the rest of the tooth (Fig 96) The fragment becomes reunited with the root by the apposition of new cementum around the fragment and on the root surface

PATHOLOGICAL RESORPTION OF TEETH

The pathological resorption of dentine and cementum occur by a mechanism similar to the resorption of bone and to the normal resorption of the deciduous teeth In the case of enamel the process is histologically similar, although its chemical nature must be different In each case demineralization and proteolysis occur simultaneously in the presence of osteoclasts which come to lie in curved indentations, Howship's lacunae, on the surface of the hard tissue

Unerupted Teeth. Teeth which have failed to erupt and remain buried in the jaws for many years very often show evidence of resorption and sometimes this is extensive The process commonly starts at the surface of the enamel which is penetrated by vascular connective tissue It is presumed that this is made possible by atrophy of the normal covering of reduced enamel epithelium When the enamel has been penetrated, the resorptive process extends in an irregular manner into the dentine but stops short of the pulp, the predentine and immediately adjacent dentine being apparently resistant to resorption (Fig 97) The boundaries of the resorbed areas show Howship's lacunae, and osteoclasts occupy these bays in places where resorption is still active The intruding connective tissue has osteogenic properties and in many resorbed areas bone is laid down in place of the lost dental hard tissues (Figs 97, 98) The process may continue for many years and large parts of the tooth may eventually be replaced by bone, at first of coarse fibre type but sometimes, later, lamellar bone with fatty marrow One result of this is that the tooth may become ankylosed to the surrounding bone being joined to it by a complicated dovetail of bone The whole process may be regarded as analogous to that which normally takes place in bone, namely continuous partial resorption and apposition In other parts of these same teeth it is common to find continuous apposition of cementum, not only on the root but also extending from the cement-enamel junction on to the enamel of the crown Since the periodontal membrane of a buried tooth is devoid of function, it tends to become thin and atrophied, and in some cases the hyperplastic cementum becomes continuous with the bone across the membrane This is a second mechanism by which a tooth may become ankylosed to the surrounding bone

Submerged Teeth Deciduous teeth which have once been erupted may subsequently come to lie buried in the bone Owing to interference with the process of continuous eruption which normally maintains these teeth in the occlusal plane, they become progressively submerged by the



Fig 97
Unerupted molar tooth in which much of the dentine of the occlusal surface has been resorbed, only a thin layer of calcified dentine and of predentine remaining over the pulp (right). Woven bone (left) has formed in the place of the dentine removed. Haematoxylin and eosin ×155

alveolar bone growing around them until they are completely buried though possibly connected with the mouth by a sinus. Permanent teeth erupting on either side of them may tend to depress them further These deciduous teeth, which in the upper jaw may come to lie close to the floor of the antrum, show not only the usual resorption of their roots but also extensive resorption of their crowns with a variable amount of replacement by bone. Both these and other teeth at the time of their removal

commonly show evidence of infection, since it is usually an inflammatory incident which leads to their discovery



FIG 98

Resorption Dentine has been deeply resorbed and on the resorbed surfaces bone has been deposited In the area formerly occupied by dentine are now fatty marrow and trabeculae of lamellar bone The latter may ankylose the tooth to the jaw Haematoxylin and eosin ×50

Erupted Teeth. Resorption of erupted permanent teeth may commence either from the periodontal membrane or from the pulp

COMMENCEMENT AT THE PERIODONTAL MEMBRANE The commonest example is apical resorption in connection with an apical granuloma following gangrene of the pulp Resulting from the liberation of toxic material through the apical canals of the tooth, the inflammatory reaction of the apical periodontal tissues includes osteoclastic resorption

of the damaged cementum and adjacent dentine of the root apex as well as the surrounding bone At a little distance from the apex



FIG. 99

Owing to heavy pressure there has been resorption of bone cementum and dentine The resorption of the tooth was still continuing at top right but elsewhere had been repaired with cementum. Van Gieson. ×95

where the stimulus may be supposed attenuated an opposite effect is observed, new cementum being deposited, sometimes in considerable thickness

Pressure of sufficient extent will cause resorption of cementum and dentine It may result from orthodontic appliances or from a buried tooth impinging on the root of an erupted tooth, or from the growth of cysts and neoplasms Gentle pressure will cause resorption of adjacent bone the cementum remaining intact, but excessive pressure causes damage to the cementoblast layer on the surface of the root

... renders the damaged surface liable to resorption In the case of ... repeated over long periods of time, much tooth substance may ... On removal of the pressure, repair with cementum follows ...)



FIG 100
Resorption of dentine which has commenced at the neck of the tooth in the periodontal membrane The normal pulp is still covered by a thin layer of dentine and predentine Haematoxylin and eosin × 13

Resorption starting at the neck of the tooth in the periodontal membrane is a well-recognized entity (Figs 100, 101) It may be the result of injury to the cementoblasts and root surface by toxic products from the gingival crevice The normal root surface is regarded as protected from resorption by an intact cementoblast layer and a continuous covering of uncalcified cementum In some cases trivial resorption is soon followed by repair with cementum In others the resorptive process burrows deeply into the dentine of the crown, hollowing it out, but respecting the dentine immediately adjoining the pulp until a very late stage Intermittent replacement of dentine with bone is usual At some stage the crown of the tooth is likely to fracture or the resorptive tissue may become infected by communication with the oral cavity, so that the pulp ultimately becomes involved

Reimplanted Teeth Teeth removed and reimplanted in the same individual may become reattached provided that the tooth surface has not been damaged by the application of drastic antiseptics, by scraping, drying, boiling, or other harsh treatment When these things have occurred, the living tissue on the surface of the root is destroyed and resorption of cementum and dentine will follow Even then the tooth may in the absence of infection be retained for some time and parts of it may become ankylosed to bone In some cases resorption will continue until little more than a root canal filling remains in the alveolar bone In the presence of infection it is more likely that the whole tooth will rapidly become exfoliated

Multiple Tooth Resorption In rare cases resorption of many erupted teeth occurs progressively and in an irregular manner by a tunnelling process commencing in the periodontal membrane in the neighbourhood of the apices. The lost dental tissue is replaced piecemeal by bone The



FIG. 101
Enlarged view × 120 of area where resorption is still active. Large darkly stained osteoclasts occupy many Howship's lacunae

pathology of this condition is obscure In affected patients various different constitutional disturbances including dysplasia of bone have been found, but rarely the same disorder in different cases and sometimes no constitutional disturbance at all has been detected It is to be supposed that there is some inherent or acquired disability in the maintenance of cementum.

More frequent are persons in whom the teeth become regularly and progressively shorter as the result of resorption from the apices but without tunnelling into the substance of the tooth The condition may occur in children and has then sometimes been ascribed to injudicious

and renders the damaged surface liable to resorption In the case of pressure repeated over long periods of time, much tooth substance may be lost On removal of the pressure, repair with cementum follows (Fig 99)



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orthodontic treatment The process is a slow one, the periodontal membrane and lamina dura being continually reconstituted, so that the histological appearance shows few abnormalities The apical ends of the teeth may become squared off, rather like the result of a successful apicectomy. Progressive shortening of the roots of many teeth is also seen in some patients with longstanding periodontal disease Here there is evidence of chronic inflammation, and toxic absorption together with increased mobility may be held responsible



FIG 102

Resorption of dentine commencing at the pulp has so hollowed out this permanent tooth that it has fractured Haematoxylin and eosin ×6

COMMENCING AT THE SURFACE OF THE PULP (Internal granuloma, pink spot) Normally the surface of the dentine next to the pulp is covered by a layer of predentine which is resistant to resorption and by a layer of odontoblasts It is probable that injury to these layers is a prerequisite for internal resorption of the dentine The injury may be the result of interruption of the blood supply of a tooth resulting from a blow, the resorption beginning when the blood supply is restored (see page 74), or it may be caused by the proximity of bacteria and their products and possibly other factors promoting an inflammatory rseponse over a long period Thus, in chronic ulcerative pulpitis it is common to find that some of the dentine next the pulp has been resorbed In certain cases, however, where the pulp is not clinically exposed, the resorption becomes so extensive that an oval cavity may occupy the whole thickness of the tooth which will become fragile and may break (Figs 102, 103) In these cases the cavity is occupied by pulp showing evidence of chronic inflammation and by proliferating granulation tissue In some parts the cavitation will be extending and osteoclasts will be seen In others the lost dentine will have been replaced by calcified matrix which is not tubular dentine or well formed bone and has been called callus by Fish (1948) (Fig 103)

HYPERPLASIA OF CEMENTUM—CEMENTOSIS

The apposition of cementum on the roots of the permanent teeth normally continues throughout life and is a part of the mechanism by

which the tooth maintains its vital attachment to the bone and by which the attachment is adjusted to the movements of the tooth in the bone. The thickness of cementum finally acquired varies considerably between individuals and may be affected by genetic factors Hyperplasia of cementum is not by itself evidence of infection. In certain circumstances



FIG. 103
Resorption had evidently ceased and a collagenous matrix had begun to be deposited at the margin of the resorbed area × 55

however apposition of cementum may be abnormally increased This may be the result of local and general disorders and may affect one or many teeth

Bacterial Stimulation. The commonest cause of cementosis is stimulation of the cementoblasts by bacterial products In the vicinity of the organisms the cementum may become necrotic at a little distance from them it is likely to become resorbed but a little further away apposition of new cementum is likely to be promoted Thus in chronic infection of

the pulp, and in chronic apical or other periodontitis, cementosis of some parts of the roots is commonly found Macroscopically evident as either a general thickening or as knob-like enlargements, the hyperplastic cementum is seen in sections to be composed of successive layers separ-



FIG 104
Hyperplasia of cellular cementum The incremental lines of Salter are seen running vertically Haematoxylin and eosin ×60

ated by resting lines, the incremental lines of Salter The cementum is of the cellular type (Fig 104) Other types of chemical stimulation may produce similar effects

Mechanical Stimulation. Excessive forces applied to a tooth will produce resorption of cementum but mechanical stimulation below a certain threshold may encourage its apposition Isolated spurs of cementum may be found extending from the tooth along groups of periodontal fibres but are uncommon

Buried Teeth. Teeth which fail to erupt very commonly develop thick layers of cementum by the time the possessor reaches middle life. This is evidence that the growth of cementum can be independent of function of the tooth The cementum may extend from the cement enamel junction



FIG. 105
Buried tooth A spur of cementum has grown over the cervical enamel which occupied the central white triangular area before decalcification. Haematoxylin and eosin. × 70

over the surface of the enamel (Fig. 105) and may also be found in the occlusal grooves The hyperplasia of cementum is not evidence of infection.

Concretion—'False Gemination' Occasionally the 2nd and 3rd upper molars become joined together by cementum uniting their roots (Fig 106) The crown of the 2nd molar is erupted but the whole of the 3rd molar remains unerupted The cause is not known but is presumed to be a mild irritation of some sort or an abnormal spatial arrangement

of the tooth germs It becomes possible because in the developmental position the roots of the 3rd upper molar are always in close proximity to those of the 2nd The condition is to be distinguished from that in which two teeth are united by dentine (page 23)



FIG 106
'False gemination' A small upper third molar (on the right) is united to the second molar (left) by a laminated mass of cementum Haematoxylin and eosin ×9

'Cementoma'—Periapical Ossifying Fibrosis. Under these names various conditions have been described. One is a spherical hard mass attached to the apex or other part of a tooth On section it has the following structure In the centre is usually the apex of a tooth covered with successive layers of cellular cementum according to the usual pattern of cementosis In the later formed peripheral layers, however, the orderly arrangement of incremental lines has been lost and apposition has become highly irregular, with the inclusion of islands of soft tissue and blood vessels (Fig 107) and the formati[illegible] isolated nodules of hard tissue Further, wherea[illegible] er for[illegible] [illegible]ue was clearly identifiable as cementum, [illegible] these special features and may well be [illegible] peripher[illegible] the mass is connective [illegible] is be [illegible] by surface appos[illegible]

The structu[illegible]
that r[illegible]

lternately in different parts of the specimen This produces a variety f mosaic pattern similar to that seen in the teeth in Paget s disease f bone the section being traversed by curved reversal lines indicating vhere resorption has ceased and apposition recommenced (Fig 108)



Fig 107

Cementoma Above is seen the dentine of the tooth apex and next to this regular cellular cementum with incremental lines. Outside this is highly irregular cementum and bone-like tissue containing vascular channels. Ground section. × 17

From radiological and histological studies it would seem that the process starts by the resorption of apical bone in relation to a vital tooth as shown by a periapical radiolucent area. This is occupied by proliferating fibrous connective tissue Within this fibrous mass hard tissue is deposited progressively while the connective tissue extends peripherally until the nodule may reach a size of one or two centimetres, after which growth ceases.

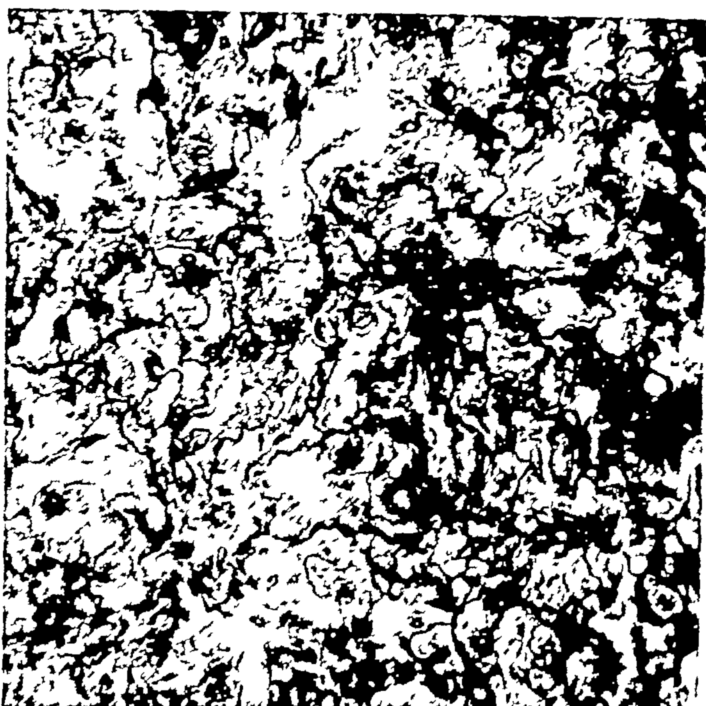


FIG 108
'Cementoma' Complicated mosaic-like pattern produced in cementum by alternating resorption and apposition Picrothionin ×50



FIG 109
Paget's disease of bone Mosaic-like pattern in the cementum of a molar tooth Picrothionin ×65

Sometimes many teeth are affected simultaneously and it appears likely that some systemic cause exists though its nature is unknown The bone is otherwise normal.

Paget's Disease of Bone Osteitis Deformans. A striking form of cementosis is seen in some elderly patients suffering from Paget's disease. The dental abnormality is only found in parts of the jaws in which the surrounding bone has been affected for many years. There is increased apposition of cellular cementum which may lead ultimately to the formation of large ragged masses around the roots Sometimes the cementosis is preceded by partial resorption of the root and throughout the process of apposition of cementum there is concurrently resorption of other parts of it, though not sufficient resorption to prevent steady increase in bulk The result of these changes is that on section the root shows a mosaic pattern analogous to that seen in the bone in this disease (Fig. 109) The later formed cementum becomes irregular in shape and character and may appear continuous with irregular masses of sclerosed bone

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4. REGRESSIVE CHANGES

When a tooth has been in use for many years it has undergone changes in shape and structure, some of which are physiological and some the result of minor injuries

Enamel. Attrition will have altered the shape of the cusps and the enamel will usually have acquired a number of cracks running from the surface to the dentine and visible in ground sections if not macroscopically These cracks come to contain organic material derived from the oral fluid or elsewhere and bacteria, as may be seen in sections decalcified with special precautions

The difference between a crack and an enamel lamella is a matter of dispute: it may be that some of the structures described as lamellae are in fact cracks

The presence of bacteria in such a crack is not by itself evidence of dental caries

Dentine. Apart from any effects of caries, the principal changes in dentine *will result from attrition and abrasion though very similar* changes will occur more slowly in the absence of attrition and even in teeth which fail to erupt

DEAD TRACTS OF FISH When the attrition reaches such a stage that the peripheral ends of dentinal tubules are exposed to the oral fluids, the odontoblasts concerned, that is those odontoblasts that have fibres lying in the tubules opened, receive stimuli which cause the sealing off of the *tubules at their pulpal ends* The effect of this is to protect the pulp It also has the result that a tract of dentine is cut off from such diffusion of fluids as normally occurs between the pulp and dentine, hence the name 'dead tract' The condition has been demonstrated by sealing dyes into the pulp chamber of teeth and cutting ground sections of the teeth after an interval of a few days In a very young tooth the dye will be found to have diffused up to the amelo-dentinal junction and perhaps slightly into the enamel In the case of a dead tract the dye does not enter the tract so that this is outlined as an unstained zone, the rest of the dentine being stained

Apart from staining by diffusion, a dead tract can often be recognised in a ground section by its opacity, appearing dark (Fig 110) in transmitted light and white in reflected light This opacity results principally from the fact that the interior of the tract is relatively inaccessible to the clearing agents used in the preparation of the section

Within the tract the dentinal fibres of the odontoblasts are degenerated and special stains will often show accumulations of fatty material in droplets The dead tract is not distinguishable in sections of decalcified material

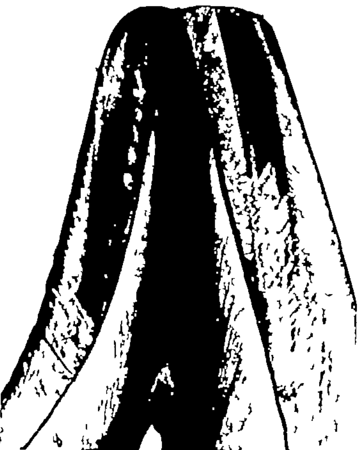


Fig. 110

(Dead tract) Ground section (×23) of a lower incisor showing dead tract of dentine running vertically between the worn incisal surface and the pulp where a little *secondary dentine has been deposited.* The dead tract appears dark by transmitted light and black in this figure



Fig. 111

Ground preparation of dentine showing tubules in cross-section and stained with silver nitrate. Lower part of figure shows a translucent zone in which the tubules are greatly narrowed or quite obliterated by thickening of the peritubular translucent area. ×530

PERIPHERAL OBLITERATION OF THE TUBULES In old teeth as seen in ground sections, the peripheral ends of the dentinal tubules become almost invisible owing to their obliteration with material of the same refractive index as the matrix of the dentine The dentine then has an appearance of rather uniform translucency This change is probably the same as that in the ‘ translucent zone reaction ’ to caries, but here occurs either as a result of age or of minor peripheral stimuli The nature of the alteration is not evident in sections of decalcified material stained by the usual methods, but can be demonstrated in ground sections cut transverse to the tubules (Blake, 1958) (Fig 111)

CRACKS are often found in the dentine either on surfaces exposed by attrition or as a continuation of cracks in the enamel They may contain organic material and bacteria and the latter may invade the dentinal tubules (Fig 112, 113).

LATE DENTINE AND SECONDARY DENTINE After the tooth has erupted and come into use, the apposition of dentine continues slowly apart from any particular stimulus such as attrition or abrasion At first this ‘ late dentine ’ is of the same structure as the dentine previously formed and its tubules are continuous with those existing already Later, the number of tubules comes to be reduced and their course may be irregular, and finally dentine matrix without tubules may be deposited These changes must be ascribed to ageing and atrophy of the odontoblasts In some parts of the tooth the apposition of tubular dentine continues longer than in others

Secondary or reactionary dentine is dentine laid down as the result of a local stimulus such as attrition Its distribution is related topographically to the area of injury, being laid down at the pulpal ends of tubules, the contents of which have been damaged or stimulated at their peripheral ends The tubules of secondary dentine, which may be numerous or few according to the severity of the injury and the age of the tooth, are usually not physiologically continuous with those of the primary dentine (Fig 114) A dye placed in the pulp will diffuse into the tubules of recent secondary dentine, but will not usually enter those of the primary dentine immediately superficial to it, which has thus become a dead tract

Cementum Age changes in cementum are the apposition of successive layers upon its surface either of the acellular or cellular variety These changes are not regressive but are concerned with the maintenance of a healthy attachment of the tooth to the bone and with its continued eruption and movement

In deciduous teeth retained abnormally long, the apposition of cementum may cease and the same may occur in permanent teeth long devoid of function or of great age

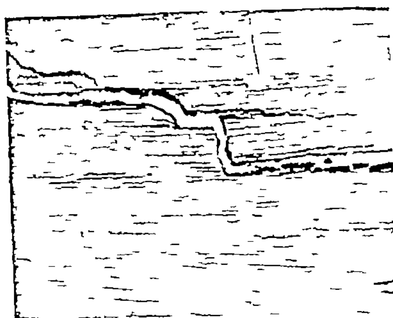


FIG. 112



FIG. 113

FIGS. 112, 113

Crack in the dentine of an incisor The space has been partly occupied by bacterial growths and some organisms have entered the dentinal tubules. (*Brit dent J* 1948) Gram's stain × 110 and × 700

The Dental Pulp. Within a few years after the tooth has entered into full function, the dental pulp may show changes from the structure usually described as normal and these changes will progress during life

OEDEMA The accumulation of extracellular fluid, indicating defects in function of pulp cells, must displace existing structures A characteristic appearance is produced in the odontoblast layer where the odonto-



FIG 114
Reactionary dentine resulting from attrition at the incisal margin Ground section ×45

blasts are crushed together by globular collections of fluid (Fig 115) ('sheaving of the odontoblasts'), and in the body of the pulp the cells and their processes become separated by similar collections of fluid so that eventually a net-like appearance is seen (Fig 116). In advanced cases many of the pulp cells are lost and the condition is described as reticular atrophy

FATTY CHANGES In ageing teeth minute droplets of stainable fatty material can be shown to appear in the pulp cells or their processes

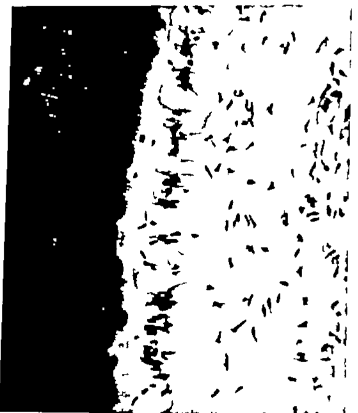


FIG. 115

Oedema of the pulp Globular collections of fluid have displaced cells in the odontoblast layer Haematoxylin and eosin. ×230



FIG. 116

FIG. 116.—Intercellular collections of fluid give the pulp a net like appearance in section Though most obvious near the odontoblasts (left) they also extend throughout the pulp Haematoxylin and eosin. ×120

This material is only seen in sections stained with special fat stains and which have not been exposed to the action of fat solvents It is not seen in sections prepared by routine methods



FIG 117
Fibrosis of pulp Section of pulp of deciduous tooth retained up to adult life The odontoblasts have disappeared and the pulp contains few cells but many collagen fibres, some arranged at right angles to the dentine In the centre the neurovascular bundle shows small deposits of calcium salts Haematoxylin and eosin ×110

FIBROSIS The young pulp contains a network of fine fibres of reticulin, as may be demonstrated in sections impregnated with silver, but does not contain fibres which take the usual stains for collagen In older teeth such fibres are found, staining pink with eosin and red with van Gieson's stain, and the number increases with age until some areas may be extensively occupied by fibrous tissue The increase in collagenous intercellular substance is likely to be accompanied by a decrease in the number of pulp cells In deciduous teeth which have been retained beyond the normal period, the whole pulp may be replaced by collagenous fibrous tissue (Fig 117)

The processes of increasing oedema and fibrosis with decreasing cellular content are sometimes described together as fibrous degeneration of the pulp The changes must be regarded as the results of ageing, of

minor local disturbances and perhaps of vascular or other disorders of systemic character

DIFFUSE CALCIFICATION A common degenerative change is the deposition of innumerable minute granules of calcific material in some



FIG. 118
Diffuse calcification of the pulp Countless small foci of calcification are scattered through the substance of the pulp Some of these have become incorporated in a large pulp stone (top right). Haematoxylin and eosin. ×55

areas of the pulp especially in the roots scattered either along existing collagen fibres or in the other intercellular substance (Fig. 118) As the deposit increases, whole fibre bundles may be involved and take a deep violet stain with haematoxylin and these elements may become aggregated into larger masses Often the fibrous sheaths of nerves are affect Whereas most regressive changes in the pulp are symptomless t change appears sometimes to be associated with paroxysmal pain wh may indicate degenerative changes in the nerve itself or in the vascu system of the pulp

SECONDARY AND LATE DENTINE The apposition of dentine continues to some extent throughout the life of the tooth and its rate is increased by local stimuli Consequently the size of the pulp becomes progressively reduced In some cases the pulp may be almost completely replaced by dentine



FIG 119
Numerous pulp stones lie free in the pulp and one is adherent to the wall of the cavity (bottom left) Many of the stones contain tubular dentine (see Fig 120) Haematoxylin and eosin ×29

FIG 120
'True' pulp stone The body consists of two denticles of tubular dentine united into a single mass by a further deposit of tubular dentine Same specimen as shown in Figure 119 Picrothionin ×230

PULP STONES These structures are composed of an organic matrix similar to that of dentine which is deposited in successive layers peripherally and calcified They may contain dentinal tubules and are then called 'true' pulp stones, but far more commonly they do not and are then called 'false' (Figs 119, 120, 121)

True pulp stones may arise from the apposition of dentine on a false pulp stone (Johnson and Bevelander, 1956)

False pulp stones are built up about a focus which may consist of a thrombus in a small vessel, dead cells or the like These provide a stimulus for the apposition of reticulin and collagen fibres and their calcification, which continue at the periphery for long periods Often

several stones will eventually coalesce and the mass produced may fill a large part of the pulp cavity

Pulp stones are classified topographically as free, adherent and interstitial (or embedded) according to their relationship to the dentine. A free pulp stone in a young pulp would as the dentine advanced become adherent and eventually interstitial that is, surrounded by the dentine and embedded in it.



FIG 121
False pulp stones of differen sizes lying free in an otherwise normal molar pulp. Haematoxylin and eosin. × 60

Microscopic pulp stones are exceedingly common in the teeth of adults and in fully formed deciduous teeth They are only evident in radiographs when large They may be particularly numerous and large and occur in young teeth in conditions where the formation of dentine matrix is severely disturbed, such as hereditary opalescent dentine and scurvy

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5. NEOPLASMS

Adamantinoma. The adamantinoma is a benign but locally invasive neoplasm of odontogenic epithelium. It becomes a central tumour of the jaws and only rarely invades the periosteum. Even allowing for the possibility that some adamantinomata of the maxilla are regarded as basal-celled carcinomata, it is a mystery why so few have been reported there. There must be considerable doubt as to whether an adamantinoma ever metastasizes, except in the manner of an aspiration to the lungs following operation. The adenocystic carcinoma, which on occasions may closely mimic the microscopic appearance of an adamantinoma, does metastasize, and no doubt it is in this way that conflicting reports have arisen.

ORIGIN. While it is agreed that the parent cell of the adamantinoma is odontogenic epithelium, it is uncertain exactly from which part of the dental apparatus the majority arise and almost all sites have their proponents.

The Enamel Organ. A vague similarity between the morphology of the enamel organ and the epithelial follicles of some adamantinomata is thought to be in favour of this site of origin. Nevertheless, as Manley (1954) points out, this similarity is only seen under the low power of the microscope and the stellate appearance of the central cells of the epithelial follicles of the adamantinoma is produced by oedema separating degenerating cells. Furthermore, Marsland (1951) has shown the stellate reticulum under normal conditions to be always associated with a stratum intermedium.

The Dental Lamina. Whereas under normal conditions no remnants of the enamel organ are left deep in the jaws, many islands of embryonic epithelial remnants of the dental lamina are scattered between the enamel organ and the surface. Manley strongly favours these cells as parent cells of adamantinomata and points out how often the alveoli of cells in the adamantinoma in the pre-cystic stage of development resemble the dental lamina.

The Epithelial Rests of Malassez. These epithelial cells have completed their function and since they readily assume the character of stratified squamous epithelium when they become hyperplastic, as in periodontal cysts, they are most unlikely to give rise to adamantinomata.

The Epithelial Lining of Periodontal and Dentigerous Cysts. While this origin is possible, it is improbable for reasons which are discussed fully under periodontal cysts.

The Oral Epithelium. Since the dental lamina develops as an ingrowth from the oral epithelium, such an origin for the adamantinoma must be a possibility. Nevertheless, since the majority of adamantinomata

remain as central lesions for many years and ulceration is rare, it must be an uncommon phenomenon It is not easy to determine in a section whether a lesion has begun in the mucosa or if it has grown up and established contact with it.

Extra-oral Epithelium Adamantinomata have been described arising in the tibia and the anterior lobe of the pituitary Since Rathke's pouch



FIG. 122
Adamantinoma Thin strands of epithelial cells separated from each other by dense bands of collagen fibres Haematoxylin and eosin. × 110

is derived from the oral ectoderm it is not so surprising that lesions similar in all respects to the adamantinoma have been described there Willis (1948) regards the so-called adamantinoma of the tibia as a bone invading, epidermoid carcinoma, but Lederer and Sinclair (1954) in their review of the literature of tibial adamantinomata suggest they are synoviomata

THE MACROSCOPICAL APPEARANCE of the adamantinoma of the jaw is quite distinctive The majority are partly solid and partly cystic, but on occasions it is completely solid The solid tissue is soft cream coloured with a fine trabecular or granular pattern on section The cysts vary greatly in size and some are filled with a semi solid brown mucinous material while others contain a clear yellow fluid The lining may be smooth and grey coloured or it may have small friable polyps The surrounding bone shows varying degrees of resorption with bony

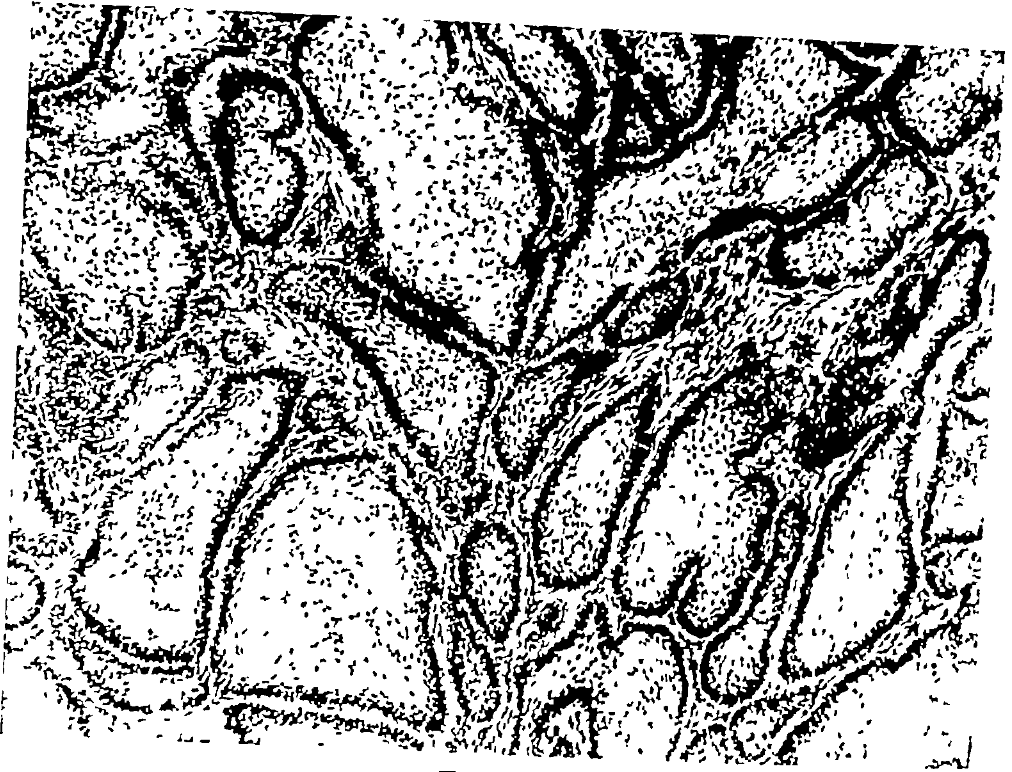


FIG 123

Adamantinoma Large trabeculae of epithelial cells, arranged with an outer rim of cubical cells and an inner mass of spheroidal cells The inner cells are undergoing various stages of degeneration Haematoxylin and eosin ×60

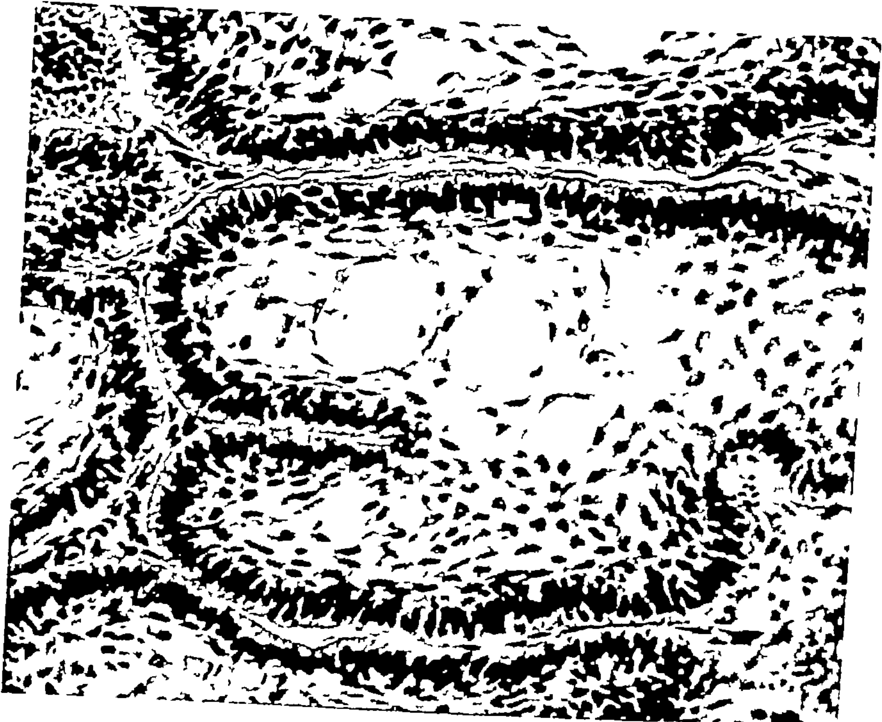


FIG 124

Adamantinoma Trabeculae of epithelial cells separated only by thin strands of fibrous tissue The central cells are degenerate and the stellate appearance is caused by intracellular oedema Haematoxylin and eosin ×220

septa surrounding some of the larger cysts The periosteum is usually intact.

The Microscopical Appearance will vary according to the degree of differentiation of the epithelium and the reaction of the stroma Thus



Fig 125

Adamantinoma. The top portion of the photomicrograph is part of the wall of a large cyst lined by a thin even layer of flattened epithelium Below, microcysts are forming in the follicles of epithelial cells. Haematoxylin and eosin. ×50

marked variation may be seen in different fields of the same tumour The epithelial cells show great pleomorphism and while in one field groups of undifferentiated cells hardly recognizable as epithelial cells because of their scanty cytoplasm, may be seen (Fig. 122) in other fields large trabeculae are found with an outer rim of tightly packed columnar or cuboidal cells and an inner mass of spheroidal cells with nuclei that are less basophilic (Fig 123) The central cells have a stellate appearance when they begin to degenerate and intercellular oedema separates one from another (Fig 124) As they degenerate the epithelial cells undergo

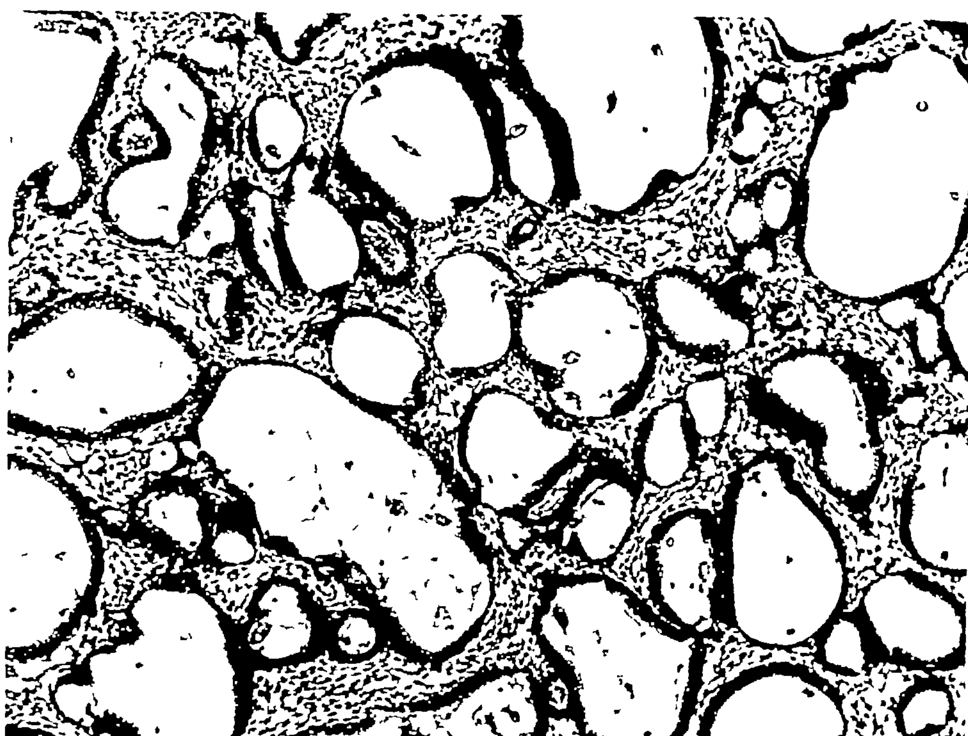


FIG 126
Adamantinoma Stromal cysts that have resulted from degeneration of the connective tissue stroma separating the follicles of epithelial cells Haematoxylin and eosin ×60

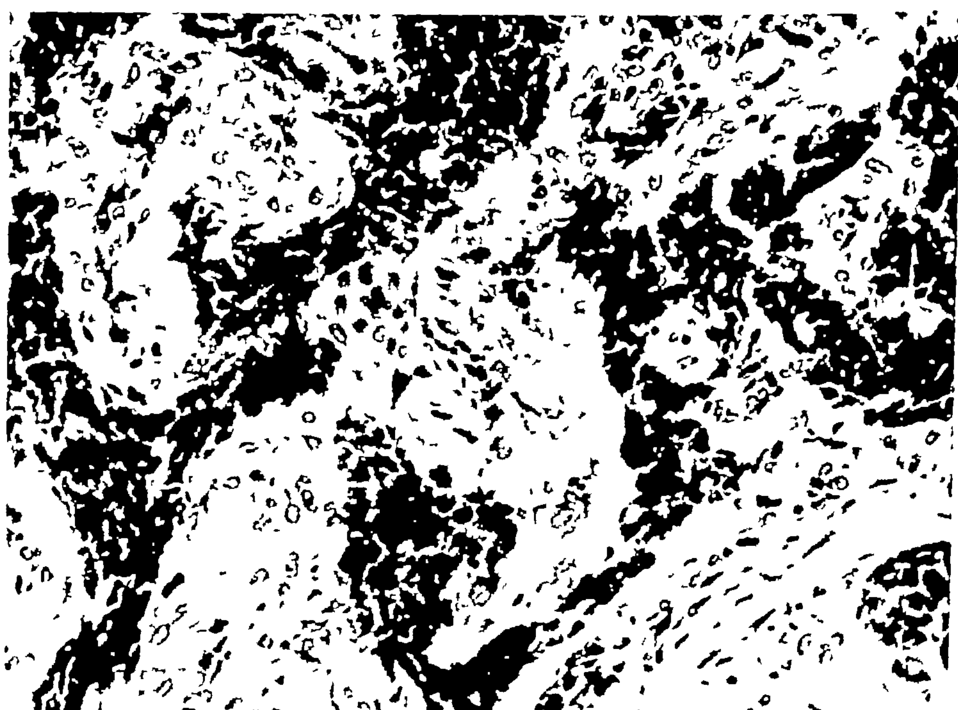


FIG 127
Melano-adamantinoma The melanin obscures the outline of the epithelial cells Haematoxylin and eosin ×200

cloudy swelling their nuclei disappear and their cytoplasm becomes markedly eosinophilic. In yet other fields of the same lesion the epithelial cells may be arranged in strands resembling the dental lamina with marginal cuboidal cells with or without intermediate cells On occasions there is differentiation towards squamous epithelium

The trabeculae of the epithelial cells are separated one from the other by fibrous tissue There may be a homogeneous zone between the columnar cells and the stroma that stains yellow with van Gieson s stain True enamel is never formed and the presence of even immature enamel or dentine should suggest that the lesion is a soft odontome rather than an adamantinoma Perhaps intermediate forms exist that show in part the features of an adamantinoma and in part those of a complex odontome

Two types of cyst formation occur the first by degeneration of the epithelial cells, the second by degeneration of the fibrous tissue Micro cysts form in the trabeculae of epithelial cells following the degeneration of the central cells and by their fusion large cysts are formed that become lined by a flattened single layer of epithelial cells, supported by fibrous tissue and sometimes bone (Fig 125) The stromal cysts form from a type of mucinous degeneration of enclaved fibrous tissue The stromal cyst is bounded by the columnar cell layer of the epithelial follicle but forms on its basal side (Fig 126)

THE MELANO-ADAMANTINOMA This rare lesion must be considered as a separate and distinct pathological entity for it has only been found in the jaws of babies under the age of four months and it would appear to be a developmental anomaly rather than a neoplasm On section there is a characteristic alveolar pattern of epithelial cells, with so much melanin that their cell outlines are often obscured (Fig. 127) While many authorities would agree that it arises from odontogenic epithelium recently MacDonald and White (1954) have demonstrated masses of neuroblasts differentiating into glial cells adjoining pigmented cubical cells all suggesting a differentiation towards a primitive retina

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11

THE PERIODONTAL TISSUES

1. DEVELOPMENTAL ANOMALIES

HEREDITARY AND IDIOPATHIC HYPERPLASIA OF THE GUMS—'FIBROMATOSIS'

In this condition there is a remarkable overgrowth of the fibrous tissue of the gums The process may start before birth but is commonly noticed during the eruption of either the first or second dentition and usually affects the whole of the gingivae, increasing with age up to adolescence but showing little further progress in the adult Some teeth may remain unerupted and others may be almost covered by the abnormal gum which characteristically shows no signs of inflammation and is firm and tough In a proportion of cases there is evidence of dominant inheritance and there may also be excessive hairiness A similar condition, affecting only the molar regions of one or both jaws, arises in late childhood and has been called 'symmetrical fibroma'

In sections the epithelial covering usually shows some hyperplasia with increased depth of the epithelial ridges and sometimes keratinization The connective tissue next to the epithelium, the lamina propria, is relatively normal and there is little evidence of inflammation except near the teeth and at any sites of injury The lamina propria is bound down to a dense mass of interlacing collagen bundles which may be several centimetres thick (Fig 128) and is continuous with the fibrous layer of the periosteum In many cases areas of mucoid change are found in this mass the collagen fibres appearing fragmented and the interstices distended with a mucoid substance

2. INFLAMMATORY REACTIONS

GINGIVITIS

Acute Gingivitis

Whether caused by physical or chemical injury or infection, in acute inflammation the gingivae will show changes similar to those observed in other soft tissues there will be hyperaemia and an exudate containing many polymorphonuclear neutrophil leucocytes In addition there may be necrosis of the epithelium and part of the underlying corium The inflammation will enter a subacute stage and may then resolve or become

chronic. When necrosis has occurred there may be a permanent loss of tissue resulting for example in shortening of the interdental papillae

Vincent's Ulcero-membranous Gingivitis. This is a special form of acute and subacute gingivitis having a characteristic clinical course and associated with a predominant flora of *Borelia vincenti* and *Fusiformis fusiformis* The aetiological rôle of these organisms is not established

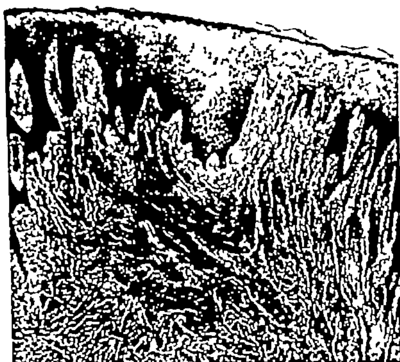


FIG. 128
Hereditary hyperplasia of the gingivae From the premaxillary region of a boy of 10 years. There is great thickening of the gingival corium which is composed of a dense mat of interlacing collagen fibres. The epithelium shows slight hyperplasia also Van Gieson. ×55

Sections (Fig 129) present a superficial necrotic coagulum consisting of the epithelium and part of the corium with great numbers of the characteristic organisms (Fig 130) fibrin and leucocytes Deep to this, the surviving corium shows acute or subacute inflammatory changes but not bacterial invasion except on the surface of the living tissue It is usually densely infiltrated with polymorphs The epithelial attachment to the tooth may be necrotic and the transeptal fibres may be eventually involved

Acute Herpetic Gingivitis is an incident in a general stomatitis due to infection with the virus of herpes simplex The histological appearances are those of acute inflammation to which may be added the special features found in other parts of the mouth (see page 141)



FIG 129
Acute ulcerative gingivitis (Vincent's) Margin of ulcer (A) Coagulum of necrotic tissue fibrin leucocytes and micro-organisms (B) Living tissue acutely inflamed and packed with polymorphonuclear leucocytes Haematoxylin and eosin ×60

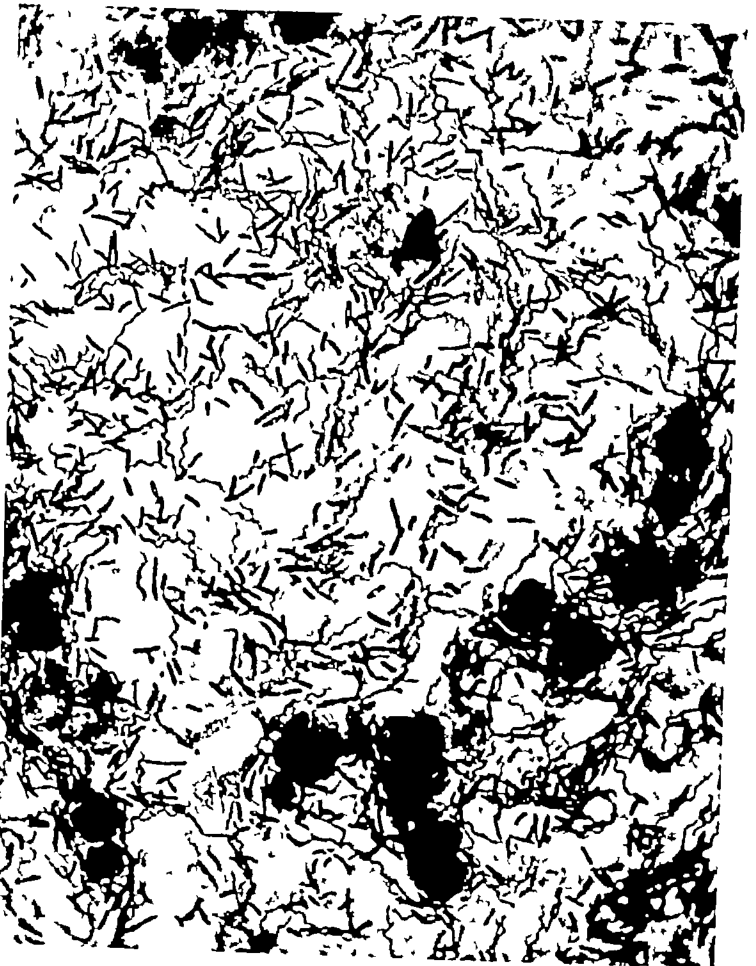


FIG 130
Smear from gum showing *Fusiformis fusiformis* and *Borelia vincenti* ×480

Untreated or unsuccessfully treated acute gingivitis may pass into the chronic stage This is characterized histologically by hyperaemia oedema and infiltration with large numbers of cells principally lympho cytes and plasma cells histiocytes and fibroblasts. Where there are

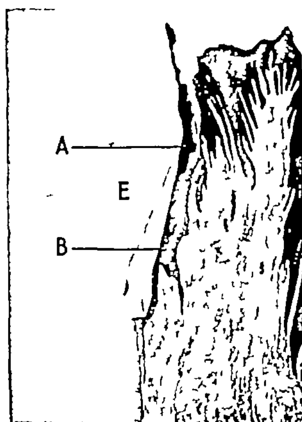


FIG. 131
Early gingivitis. Calculus (A) is attached to the enamel cuticle as deep as the level of the gingival crevice At this point the epithelial attachment is very thin and beneath this point it has begun to proliferate (B). The deeper part of the epithelial attachment is still thin and regular It extends on to the cementum. There is a moderate infiltration with lymphocytes and plasma cells between the vertically arranged fibres of the interdental papilla Part of this results from the more advanced gingivitis at the adjacent tooth The enamel space (E) contains a displaced portion of enamel matrix. Haematoxylin and eosin ×29

ulcerated areas for example in contact with masses of calculus the surface of these areas is composed of granulation tissue with many poly morphonuclear leucocytes The surface of the surrounding epithelium will not be keratinized and in parts the total width of the epithelium will be very small. Numerous projections from the deep surface of the epithelium will, however have extended far into the corium (Fig 131)

A section of the most normal human gum which can be found will show some evidence of chronic inflammation in the form of collections of lymphocytes and plasma cells and even polymorphonuclear leucocytes, since the gingivae are always subject to mechanical and chemical stimuli

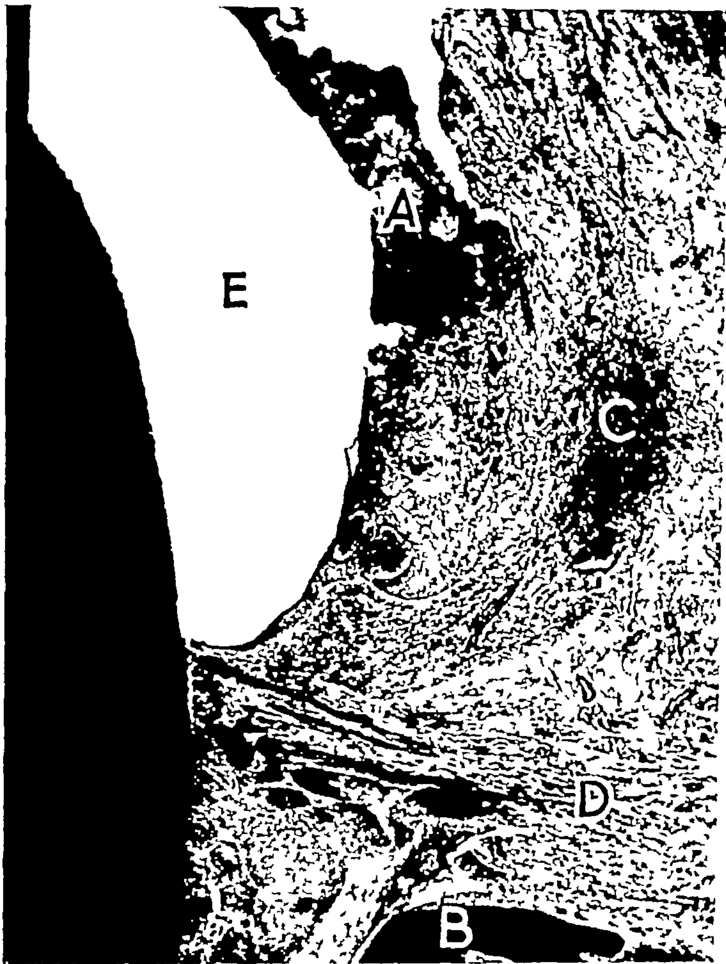


FIG 132

Chronic gingivitis Distal surface of lower first molar at 14 years There is a deposit of calculus and bacterial plaque (A) on the enamel (E) at the gingival crevice and the adjacent gingiva is ulcerated There is a dense infiltration with lymphocytes at (C) and the epithelial attachment shows irregular proliferation. The transeptal fibres (D) and alveolar crest (B) are intact Light green, fuchsin and iron haematoxylin × 27.

of many kinds The difference between e clinically norn ues and those in gingivitis is in this respect* gree Like rough the deep surface of the epithelial will bec hat irregular in clinically normal tissu is its will become extreme

The commonest form of chronic gingivitis is that associated with defective oral hygiene. In an early case (Fig 131 132) the evidence of inflammation will be related to the gingival crevice where there is often a deposit of calculus on the enamel Deep to this point the epithelial attachment may still have the normal form of a narrow band but opposite the crevice it will be thinned or ulcerated and show a branched structure with downgrowths into the corium At the level of the crevice will be a collection of lymphocytes and plasma cells in the corium and these will extend in an apical direction but not deep to the level of the transeptal fibres Between the calculus and the gingiva an exudate of polymorphonuclear leucocytes is often found

In chronic hyperplastic gingivitis more common in children the gingivae are enlarged by both hyperaemia and oedema and also by the production of new fibrous connective tissue Inflammatory hyperplasia may also produce a local pedunculated swelling on the gums an epulis (see page 121)

Calculus In sections of gingival and periodontal tissues salivary calculus and bacterial plaque are seen

Bacterial plaque is an uncalcified deposit or growth consisting principally of masses of micro-organisms of many kinds attached to the dental cuticle or to surfaces of enamel cementum or dentine It appears in preparations stained with haematoxylin and eosin as a purple layer the outer margin of which has a fringe like character The bacterial plaque associated with caries and that associated with the formation of calculus is not visibly different.

Calculus appears in decalcified preparations, stained in the same way as a bulky deposit on the dental cuticle or on the surface of the dental hard tissues (Fig 133) Its surface is usually uncalcified and is composed of bacterial plaque Its deeper parts have been calcified and it is often possible to see that it has been built up by the superimposition of layers in a rather irregular fashion presumably by the calcification of successive layers of bacterial plaque The calcified parts of the calculus usually stain less deeply with haematoxylin than the uncalcified surface

In sections where calculus and gingivae can be seen in undisturbed relationships (Figs. 131 132) the surface of the gum is usually in contact with the uncalcified bacterial plaque on the surface of the calculus or is separated from this only by necrotic cells and leucocytes. It shows evidence of chronic inflammation including often ulceration Shrinkage in the preparation of specimens often produces an artificial gap between surfaces originally in contact.

Certain intoxications or diseases and even physiological states such as pregnancy predispose to gingivitis, which may have special characteristics



Fig 133

Decalcified section of supragingival calculus attached at the neck of a tooth (A) Previously calcified material formed of irregular superimposed layers (B) Deeply stained surface layer of bacterial growths not calcified Haematoxylin and eosin ×65

Dilantin (Phenytoin Sodium B P) Following the prolonged administration of sodium diphenylhydantoinate, a characteristic gingival enlargement may be produced, perhaps by a modification of the tissue response to local injury The principal histological feature is great increase in the thickness of the corium which is composed of dense interlacing fibrous bundles Evidence of inflammation may in many parts be slight There is a tendency for the epithelium to be well keratinized and to show

acanthosis, and there may be some keratinization at the deepest parts of some of the epithelial ridges in the form of concentric cell nests

Metals. When lead or bismuth has been administered some particles of these metals in the form of insoluble salts are often deposited



FIG. 134

Bismuth line Irregular black particles of metallic salt have been deposited in the tissue between the capillary wall and the basal layer of the epithelium The capillary is seen running vertically up the centre of the figure. Haematoxylin and eosin. ×490

in the gum in the vicinity of the capillaries of the connective tissue papillae lying between the epithelial ridges. The quantity may be sufficient to be visible clinically as a blue or grey line especially in stagnation areas and mouths where oral hygiene has been poor It is assumed that chemical products of decomposition lead to the precipitation of metallic salts in the tissues (Fig 134) In mercury poisoning, ulceration may be promoted

Foreign Body Granuloma. In some cases of chronic gingivitis there is evidence of reaction to foreign particles such as silica which have become implanted in the gingivae. There are numerous giant cells,



FIG 135

Foreign body granuloma of the gum. Numerous foreign body giant cells in an area of chronic inflammation of the corium. Examination with the polarizing microscope showed birefringent crystals probably silica within and amongst the giant cells. Haematoxylin and eosin × 120



FIG 136

Foreign body granuloma is seen with the polarizing microscope. Two doubly refracting crystalline particles with giant cells, fibroblasts and lymphocytes. Haematoxylin and eosin × 190

derived from the fusion of macrophages, some of which contain the foreign particles areas of granulation tissue fibrosis and hyaline collagenous deposits (Fig 135) In the case of silica the particles may be demonstrated by their property of birefringence when a polarizing

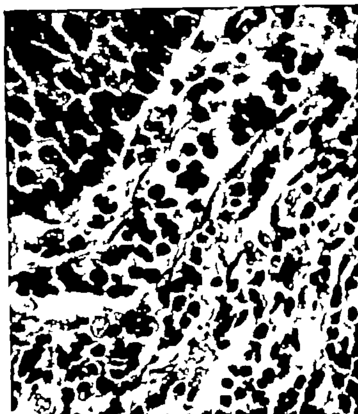


FIG. 137
Leukaemia. Section of gingiva showing a blood vessel just beneath the epithelium (top left) packed with abnormal leucocytes. These have also infiltrated the corium. Haematoxylin and eosin. ×490

microscope is used (Fig. 136) In the case of buried pieces of amalgam the reaction of the gingival tissues is very slight but macrophages loaded with particles of silver salts may be seen migrating from the area.

Scurvy The gingivitis of scurvy is notorious but the histological appearances are rarely sufficiently characteristic to be recognized as diagnostic in human material Oedema, haemorrhages and degenerative changes in collagenous structures are found in addition to chronic inflammatory changes.

Leukaemia. In leukaemias the connective tissue of the gums is often heavily infiltrated with abnormal leucocytes The lumen of capillaries and the surrounding tissue may be packed with these cells (Fig 137) vessels may be thrombosed and there may be areas of necrosis and ulceration



FIG 138
Characteristic of gingival enlargement associated with pregnancy are dilatation of capillaries and oedema sometimes without much cellular infiltration Haematoxylin and eosin ×65





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Pregnancy In the gingivitis associated with pregnancy a characteristic feature is the great vascularity of the tissues in which capillary vessels are very numerous and often dilated into wide thin walled blood spaces (Fig 138)

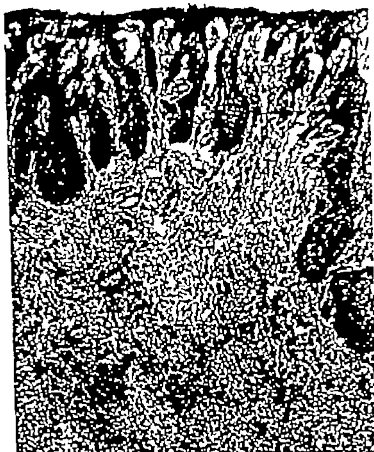


FIG. 140
Fibrous epulis with dilated capillaries adjacent to the hyperplastic epithelium, and diffusely infiltrated with chronic inflammatory cells. Haematoxylin and eosin × 70

EPULIS AND FIBRO EPITHELIAL POLYP

The **Fibrous Epulis** is a localized chronic inflammatory hyperplasia of the gum a response to chronic irritation (Fig. 139) The histological picture varies as it does with all lesions that are inflammatory in origin, according to whether or not the irritant is still evoking an active inflammatory response at the time of examination All gradations are seen between a vascular oedematous fibrous tissue diffusely infiltrated with lymphocytes and plasma cells, supporting an inflamed hyperplastic stratified squamous epithelium (Fig 140) and a relatively avascular mass of collagen fibres supporting a well keratinized epithelium (Fig 141) Since the epulis arises from the parosteal tissues which possess



Fig 141

Fibrous epulis that is less vascular than the one illustrated in Figure 140, but still containing chronic inflammatory cells dispersed between the collagen fibres Haematoxylin and eosin ×60



Fig 142

Fibrous epulis with woven bone formation Haematoxylin and eosin ×61

great osteogenic potentialities, some degree of bone formation is often present. This bone formation varies from coarse fibred woven bone to round calcific haematoxyphil deposits (Fig. 142)

The **Giant Cell Epulis** is in the nature of excessive and aberrant osteogenic granulation tissue, resulting from hyperplasia of the alveolar muco-periosteum. The great osteoclastic capacity of the alveolus is

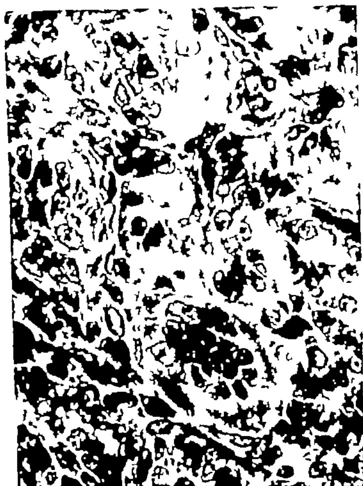


FIG 143

The giant-cells and the stromal cells in a giant-cell epulis. Haematoxylin and eosin. × 490

demonstrated by the resorption of deciduous teeth the modelling resorption of the alveolus during the mixed dentitional period and the rate at which the alveolus is resorbed when rendered edentulous Chronic irritation from calculus or the trauma of an extraction may initiate hyperplasia in which osteoclasts are produced far in excess of what would commonly result from the original exciting stimulus and persist after the stimulus has gone. The term peripheral osteoclastoma for the lesion

is an unfortunate one, since it tends to link this hyperplasia with neoplasms of the long bones

There are two main cell types that give to the giant cell epulis a distinctive microscopical appearance The first are multinucleated giant cells with 2 - 30 centrally placed nuclei and indistinguishable from osteoclasts, which are diffusely scattered through the lesion, or arranged focally The second are conveniently referred to as stromal cells They vary from polygonal forms condensed into sheets to well differentiated fibroblasts separated by variable amounts of collagen fibres (Fig 143) There is a rich supply of capillary blood vessels and haemosiderin pigment is often present In the fibrous areas the giant cells are sparse and coarse-fibred woven bone is observed The covering layer of stratified squamous epithelium is separated from the giant cells by a layer of subepithelial fibrosis (Fig 144)

All gradations are seen between the young lesion with immature polygonal cells, many capillaries and giant cells on the one hand, and older lesions that are relatively avascular, fibrotic and containing areas of woven bone on the other There is never any capsule

The **Fibro-epithelial Polyp** is simply reparative scar tissue Many polyps removed from the cheeks, lips, tongue or gum show a similar histological picture of thick bundles of collagen fibres criss-crossing each other without special arrangement, and supporting a thin layer of well-keratinized stratified squamous epithelium This appearance could result from direct trauma such as a bite to the tip of the tongue or lip, while on the gum it could represent either the final condition of a fibrous or giant cell epulis Although this appearance may resemble a fibroma, as does all other scar tissue, there is no evidence that it is a neoplasm and the term fibro-epithelial polyp is used to suggest its non-neoplastic reparative nature (Fig 145)

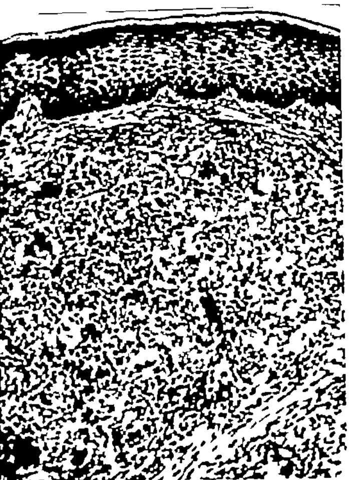


FIG. 144

Giant-cell epulis with the covering epithelium separated from the giant-cells by a layer of subepithelial fibrosis Haematoxylin and eosin ×120

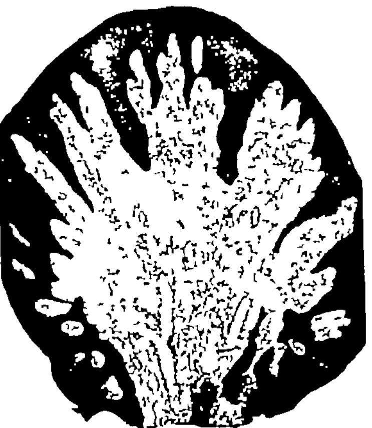


FIG. 145

A small fibro-epithelial polyp, consisting of fibrous tissue covered with stratified squamous epithelium Haematoxylin and eosin. ×46

PERIODONTITIS

In most cases periodontitis is a sequel to or concomitant of gingivitis but some forms of local periodontitis occur independently, for example the apical periodontitis associated with infection and gangrene of the dental pulp (see page 57), and these will not be discussed here The distinction between gingivitis and periodontitis rests upon some evidence that periodontal structures other than the gum are involved, particularly the alveolar bone

Acute Periodontitis

Apart from apical periodontitis, acute inflammation of the periodontal membrane is most likely to be the result of mechanical injury such as a blow to the tooth or a perforating wound from a bristle, bone splinter, reamer, or the like Human material showing the acute stage is rarely available for section, but one would see the usual changes found in acute inflammation of other connective tissues, with evidence also of mechanical injury in the form of lacerated and crushed tissues, haemorrhage, and possibly fractures of bone and cementum and foreign bodies Within a day or so resorption of damaged surfaces of hard tissues will have commenced

An acute periodontal abscess other than an apical abscess may result from an infected penetrating wound but is more commonly a complication of chronic periodontitis and will be considered in that connection

Chronic Periodontitis

Chronic periodontitis is commonly the sequel to chronic gingivitis and is recognized histologically by evidence of damage to the periodontal tissues, and particularly the alveolar bone, associated with signs of chronic inflammation

In assessing the extent of alteration of the periodontal structures likely to be due to disease, the age of the patient must be taken into account The position of the epithelial attachment on the surface of the tooth normally changes with age, moving very slowly from the enamel in an apical direction and being correlated with the continued eruption of the tooth which is regarded as a biological compensation for attrition The presence of the whole epithelial attachment on the cementum of a young tooth would thus be an indication of disease, whereas the same position on an older tooth would not be

In an early [illegible] of chronic periodontitis the following changes may be seen The epithelial attachment may be partly on the enamel and

partly on the cementum (Fig. 146) There will probably be calculus and bacterial growths on the surface of the enamel and in contact with the gingiva (Fig. 147) and at this point the latter will show infiltration with cells, deep proliferation of epithelium and possibly ulceration The

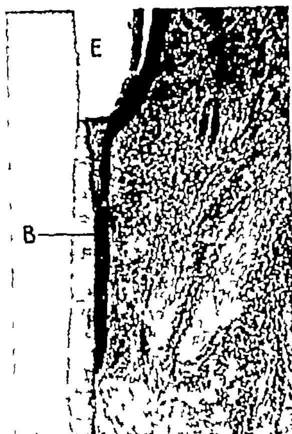


FIG. 146
Early periodontitis. The epithelial attachment (B) is partly on the enamel partly on the cementum. There is an infiltration of plasma cells and lymphocytes between the connective tissue fibre bundles and on the side of the epithelial attachment away from the tooth, an area from which collagen fibres have disappeared. There are some fragments of enamel matrix in the space (E) left by the enamel dissolved in preparation of the section. Haematoxylin and eosin, × 80

attachment of many collagen fibres including transeptal fibres to the cementum at the neck of the tooth will have been lost, the site of their former attachment being now occupied by the epithelial attachment (Fig 146) and the fibre bundles will be partly disrupted and separated by collections of plasma cells and lymphocytes New fibres will be forming at a deeper level. Resorption of the bone of the alveolar crest

will have commenced (Figs 147, 148) These changes are believed to be the response of the tissues to the entry of toxic substances through the damaged epithelial surface and do not indicate an invasion of bacteria into the gum or periodontal structures Cementoblasts and fibroblasts

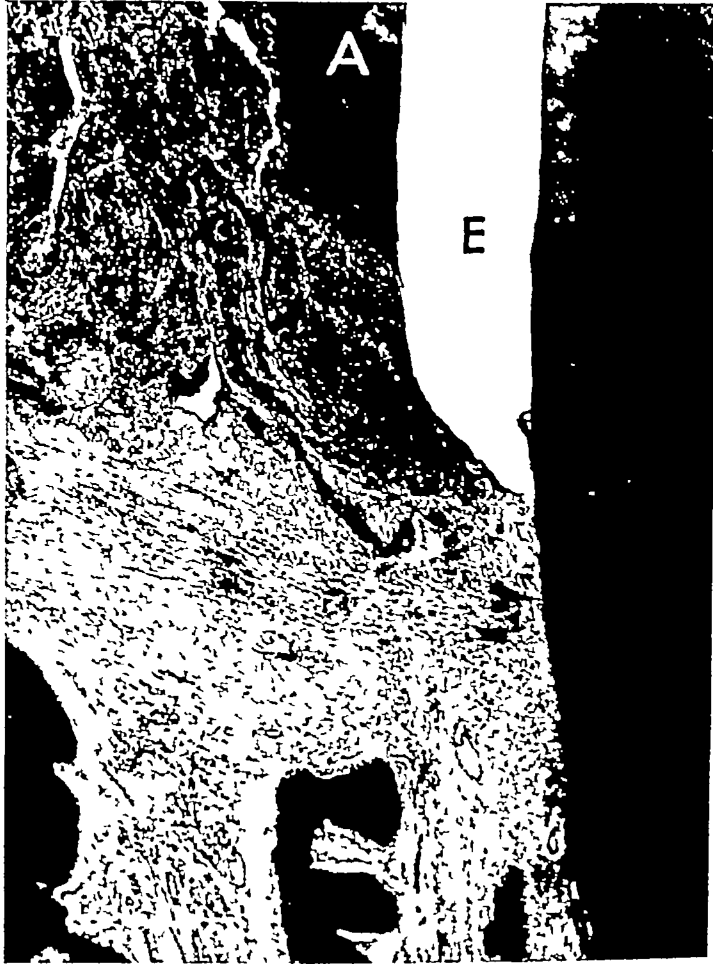


FIG 147
Early periodontitis Calculus and bacterial plaque (A) are situated on the enamel (E) and are in contact with the gingiva which is chronically inflamed The epithelial attachment is partly on the cementum but has been pulled away slightly in preparation Resorption of the alveolar crest has begun (see Fig 148) Haematoxylin and eosin ×27

are damaged and collagen fibres disappear probably through the mediation of proteolytic enzymes

The epithelial attachment continues to grow in an apical direction at a rate faster than normal and comes to lie wholly on the cementum Its too rapid growth is stimulated by toxic and mechanical factors, and its advance along the surface of the tooth is made possible by cessation of cementum production and disintegration of the attached collagen fibres

immediately deep to it The surface of the epithelial attachment applied to the cementum is composed of the older cells except at its deepest part and may have the appearance of a homogeneous keratinized layer or cuticle As the gingival crevice also moves in an apical direction



Fig 148
An adjacent section to that shown in Figure 147 Resorption of bone has commenced at (B) and there is cellular infiltration along the course of vessels at (C) and between the transeptal fibres (D). Light green fuchsin, and iron haematoxylin × 33

this layer of older or keratinized cells remains attached to the cementum while the rest of the epithelial attachment, including its basal layer becomes separated from the tooth and forms the gingival wall of a pocket.

At this stage there will be the following appearances. On the cementum at the neck of the tooth there will be calculus partly supra gingival and partly subgingival. The subgingival calculus will probably be separated from the gingival epithelium by uncalcified bacterial growths



FIG 149
Periodontitis On the left, resorption of the crest of the alveolar process has begun and several giant cell osteoclasts may be seen in Howship's lacunae To the right of this is the periodontal membrane and a root upon which the apposition of cellular cementum is continuing Haematoxylin and eosin × 120



FIG 150
Periodontitis Resorption of bone commencing within the marrow spaces of the alveolar process The marrow is fibrous and deeply stained giant cell osteoclasts are present Haematoxylin and eosin × 120

and cellular inflammatory exudate The gingival epithelium may be ulcerated on the surface which is applied to the calculus and the gingival corium will be densely infiltrated with cellular exudate and hyperaemic. The original transeptal fibres will have been destroyed and the new ones formed at a deeper level will have suffered damage and will be separated by collections of plasma cells and lymphocytes. Similar collections will be found along the course of blood vessels approaching and leaving the area through the alveolar bone and in the region of the epithelial attachment.

The crest of the alveolar bone will have undergone resorption and Howship's lacunae and osteoclasts will be found (Fig. 149) In addition, resorption may be occurring at the margins of marrow spaces not immediately on the surface of the bone (Fig. 150) At other sites further from the inflamed area new bone may be in course of apposition Fatty marrow is replaced by fibrous marrow

These processes may continue until the bottom of the pocket approaches the apex of the tooth (Figs 151 152) but their extent frequently differs greatly on different aspects of the same tooth

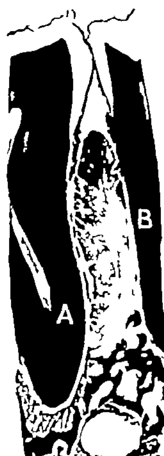


FIG 151

Chronic periodontitis, advanced. Pockets extend as far as (A) on the 1st molar and to (B) on the 2nd premolar Bone (black) has been resorbed almost to the apices of the teeth The pockets contain calculus and bacterial growths, and their gingival walls consist of chronically inflamed and partly ulcerated epithelium and granulation tissue Light green fuchsin and iron haematoxylin ×5

Periodontal Abscess. As well as resulting from an acute stimulus such as an infected wound, a periodontal abscess is frequently a complication of chronic periodontitis In such a case histological preparations will show evidence of chronic periodontitis, probably with an ulcerated periodontal pocket (Fig. 153) from which bacteria have entered the periodontium as a result of mechanical interference and tooth movement. The formation of a haematoma and the introduction of foreign particles such as calculus may have aided the micro-organisms to establish themselves in the tissues leading to an intense inflammatory

response and the formation of pus The fibre structure of the periodontal membrane is locally destroyed The abscess will be limited by condensed connective tissue, fibrin deposit and leucocytes, and the cementoblasts on the surface of the adjacent root will probably have been destroyed The adjacent alveolar bone will show resorption At a later stage the



FIG 152

Advanced periodontitis Half way down the deeper pocket shown in Figure 151 The calculus has become accidentally detached from the surface of the tooth on the left The gingival wall of the pocket is almost bare of epithelium and is densely infiltrated with leucocytes The epithelium has proliferated deeply into the interdental connective tissues Light green, fuchsin and iron haematoxylin ×60

abscess will have discharged into the base of the pocket or elsewhere and a sinus will exist

Periodontitis is called *simplex* or *complex* according to is regarded as principally the result of local irritating facto factors with the addition of important systemic factors Fi tissues taken from a boy with diabetes mellitus and is periodontitis complex

3. DEGENERATIVE CHANGES

PERIODONTOSIS

While cases in which systemic factor important influence on the progress of peri rare cases have been reported also whe extensive degenerative and destructive ch in which evidence of inflammation has has been called periodontosis and is re

degenerative change possibly representing an inability of the patient's tissues to repair by the formation of new collagenous structures the repeated minor injuries always experienced and the losses occurring from physiological resorption Or it may be the result of a lowered threshold for common stimuli which cause resorption, or in some other respect a failure of maintenance of the supporting structures of the teeth Loosening and migration of the teeth in the absence of pocket formation are regarded as characteristic of the early clinical findings.

No one medical disorder has been associated with periodontosis and it may be that any condition in which there is interference with the capacity to form collagenous matrix can be a cause of such conditions avitaminosis C protein deficiency and disorders of adrenal steroid production are examples

The histological findings have included resorption of both alveolar bone and of parts of the roots replacement of the periodontal fibres by loose vascular connective tissue without orientation and widening of the periodontal membrane These changes may occur first around the apical half of the root or only on one side of it and the tooth will migrate away from the side which has changed. A pocket will eventually develop on this side also



FIG. 153

Periodontal abscess. An abscess (A) lies just superficial to the crest of the alveolar bone, the adjacent surface of which is beginning to be resorbed (B). The gingival margin is ulcerated where it has been in contact with bacterial plaque and calculus seen slightly displaced above The abscess was discharging at this point by means of a sinus not here shown Light green fuchsin and iron haematoxylin ×25

The condition has been more frequently recognized in adolescents and young adults. In older patients a co-existing periodontitis would be likely to mask the condition and a diagnosis of periodontitis complex would be made

response and the formation of pus The fibre structure of the periodontal membrane is locally destroyed The abscess will be limited by condensed connective tissue, fibrin deposit and leucocytes, and the cementoblasts on the surface of the adjacent root will probably have been destroyed The adjacent alveolar bone will show resorption At a later stage the



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Advanced periodontitis Half way down the deeper pocket shown in Figure 151 The calculus has become accidentally detached from the surface of the tooth on the left The gingival wall of the pocket is almost bare of epithelium and is densely infiltrated with leucocytes The epithelium has proliferated deeply into the interdental connective tissues Light green, fuchsin and iron haematoxylin ×60

abscess will have discharged into the base of the pocket or elsewhere and a sinus will exist

Periodontitis is called *simplex* or *complex* according to whether it is regarded as principally the result of local irritating factors or of local factors with the addition of important systemic factors Figure 151 shows tissues taken from a boy with diabetes mellitus and is an example of periodontitis complex

3. DEGENERATIVE CHANGES

PERIODONTOSIS

While cases in which systemic factors are thought to have an important influence on the progress of periodontitis are not uncommon, rare cases have been reported also where there has been evidence of extensive degenerative and destructive changes in the periodontium but in which evidence of inflammation has been minimal This condition has been called periodontosis and is regarded as a non-inflammatory

degenerative change possibly representing an inability of the patient's tissues to repair by the formation of new collagenous structures the repeated minor injuries always experienced and the losses occurring from physiological resorption Or it may be the result of a lowered threshold for common stimuli which cause resorption or in some other respect a failure of maintenance of the supporting structures of the teeth Loosening and migration of the teeth in the absence of pocket formation are regarded as characteristic of the early clinical findings



FIG. 153

Periodontal abscess. An abscess (A) lies just superficial to the crest of the alveolar bone the adjacent surface of which is beginning to be resorbed (B). The gingival margin is ulcerated where it has been in contact with bacterial plaque and calculus seen slightly displaced above The abscess was discharging at this point by means of a sinus not here shown. Light green fuchsin and iron haematoxylin × 25

No one medical disorder has been associated with periodontosis and it may be that any condition in which there is interference with the capacity to form collagenous matrix can be a cause of such conditions avitaminosis C protein deficiency and disorders of adrenal steroid production are examples

The histological findings have included resorption of both alveolar bone and of parts of the roots, replacement of the periodontal fibres by loose vascular connective tissue without orientation and widening of the periodontal membrane These changes may occur first around the apical half of the root or only on one side of it and the tooth will migrate away from the side which has changed. A pocket will eventually develop on this side also

The condition has been more frequently recognized in adolescents and young adults In older patients a co-existing periodontitis would be likely to mask the condition and a diagnosis of periodontitis complex would be made

Atrophy of the Periodontium, whether senile, pre-senile, or resulting from earlier inflammatory changes, is characterized histologically by quantitative differences, there being a decrease in the intra-alveolar proportion of the tooth, in the thickness of the gingivae, in the number of periodontal fibres, in the number and thickness of bone trabeculae and in the thickness of the periodontal membrane

When a tooth is disused the periodontal membrane also becomes thinner and loses the characteristic arrangement of fibres which no longer enter the bone and cementum as Sharpey's fibres

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III

THE ORAL MUCOSA

1 REACTIONS OF THE ORAL MUCOSA

The reactions of squamous epithelium are very limited a prickle cell can form keratin or it can divide The following terms are used to denote microscopical appearances in the epithelium —

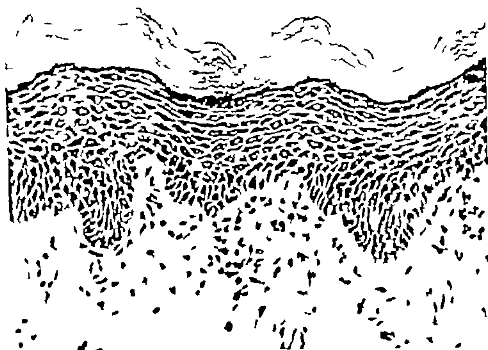


FIG. 154

Keratosis of the buccal mucosa with a well-defined keratin and granular layer Haematoxylin and eosin ×260.

Hyperkeratosis and Keratosis. Hyperkeratosis is an increase in the thickness of the keratin layer but since the oral mucosa other than the masticatory mucosa is not normally keratinized the formation of an abnormal keratin layer is more suitably referred to as keratosis The granular layer is pronounced but the prickle cell layer may be normal in thickness (Fig. 154)

Parakeratosis. Whereas in hyperkeratosis the stratum corneum is perfectly cornified in parakeratosis is it imperfectly cornified and the

nuclei of the cells remain (Fig 155) This change is commonly associated with intercellular and intracellular oedema of the prickle cell layer, which appear to interfere with the normal formation of a granular layer.

Acanthosis is an increase in width of the prickle cell layer While it may accompany the increased formation of keratin in leucoplakia, it

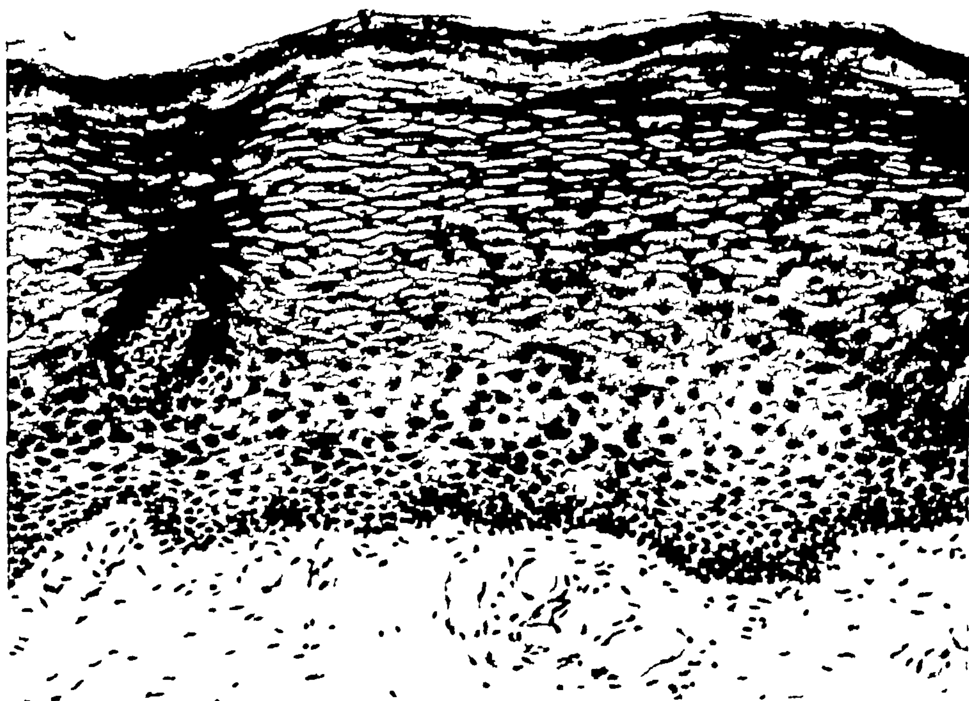


FIG 155
Parakeratosis of the buccal mucosa Haematoxylin and eosin ×145

may occur alone as it does in the non-keratinized papilloma This proliferation results in a broadening and lengthening of the epithelial ridges (Fig 156)

Spongiosis is intercellular oedema of the prickle cell layer, and often predisposes to parakeratosis There may be an infiltration of polymorphonuclear leucocytes and lymphocytes between the prickle cells The protoplasmic bridges between the prickle cells are clearly visible (Fig 157)

Intracellular Oedema is often associated with intercellular oedema and gives rise to vacuolated cells (Fig 158)

Vesicle and Bulla. The difference between these two is only one of size They are collections of fluid between the epithelial cells and may arise in two ways

1 Intercellular oedema may be such that the intercellular protoplasmic bands of the prickle cells are parted, resulting in separation of

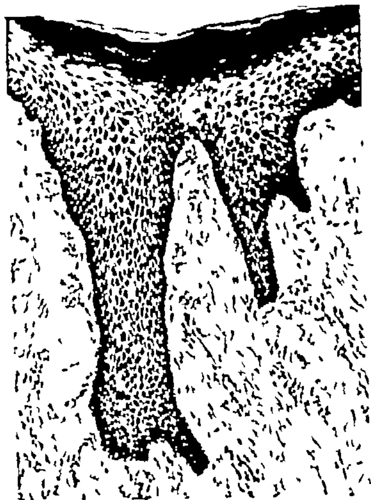


Fig. 156
Acanthosis of the buccal mucosa. Haematoxylin and eosin × 120

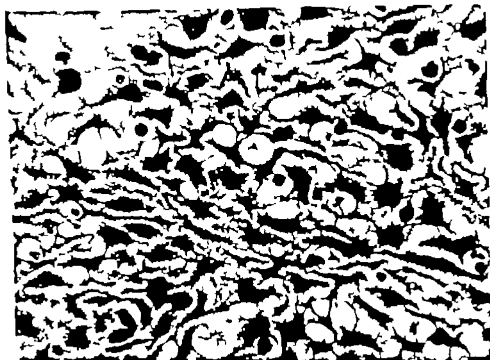


Fig. 157
Spongiosis of the oral mucosa. The intercellular protoplasmic bridges between the prickle cells are clearly visible. A few polymorphonuclear leucocytes and lymphocytes have infiltrated between the prickle cells. Haematoxylin and eosin. × 580

the cells This procedure is accentuated if there is an associated lack of resistance on the part of the intercellular bridges, a condition known as acantholysis, such as may be found in pemphigus

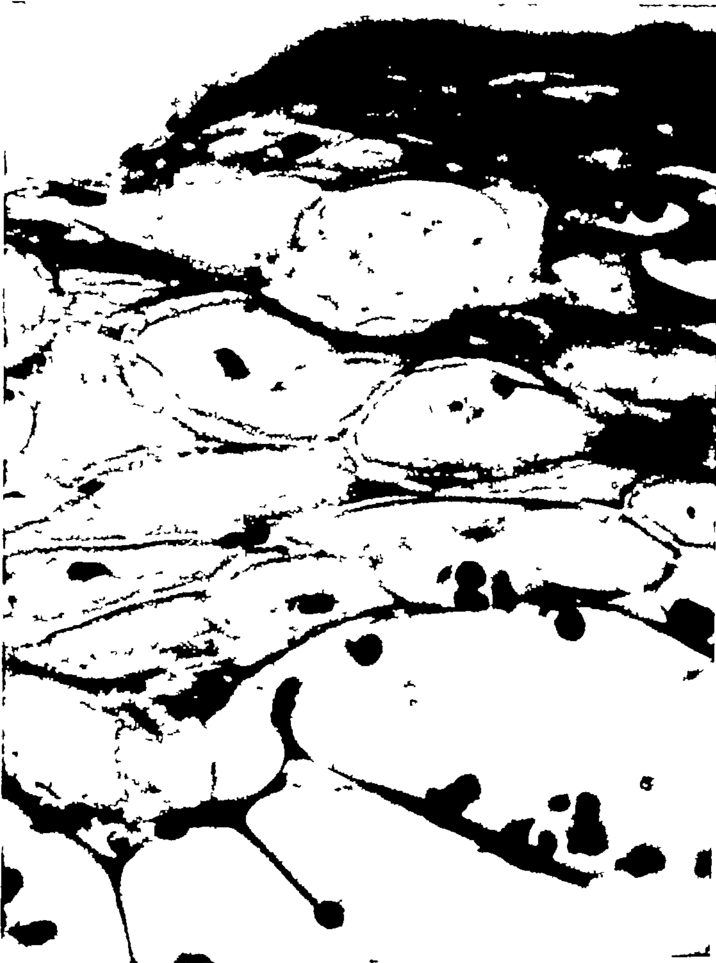


FIG 158
Intracellular oedema of the buccal mucosa, with vesicle formation at the bottom of the picture Haematoxylin and eosin ×500

2 The epithelial cells may themselves degenerate and the empty swollen cells form an open meshed reticulum This type of degeneration, known as reticular degeneration, is seen in infections with the herpes simplex virus The vesicle increases in size with the breakdown of adjoining cell membranes (Fig 165)

2. DEVELOPMENTAL ANOMALIES

Fordyce Spots. Buff-coloured shotty macules are commonly observed on the oral mucosa, especially around the opening of Stenson's duct and the lips These spots are due to the presence of sebaceous glands and have no clinical importance The glands are of the multiple acinar type

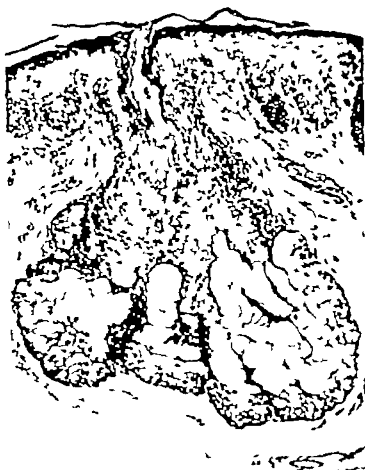


Fig. 159

Sebaceous gland and its duct in the mucosa of the vermilion border of the lip. Haematoxylin and eosin ×105

and their ducts open directly on to the surface of the oral mucosa (Fig 159) unlike the majority on the skin which are appendages of the outer root sheath of the hair follicle

The Oral Epithelial Naevus. This is a malformation either present at birth or developing later in life in which hyperplasia of the prickle cells occurs without the presence of ordinary (dopa positive) naevus cells. Developmental anomalies of the oral mucosa occur either alone or in

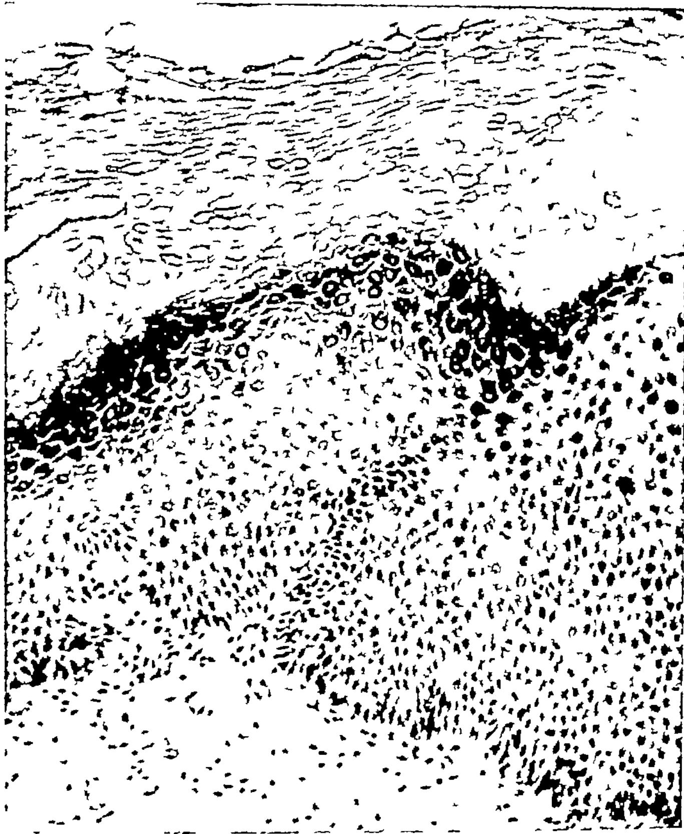
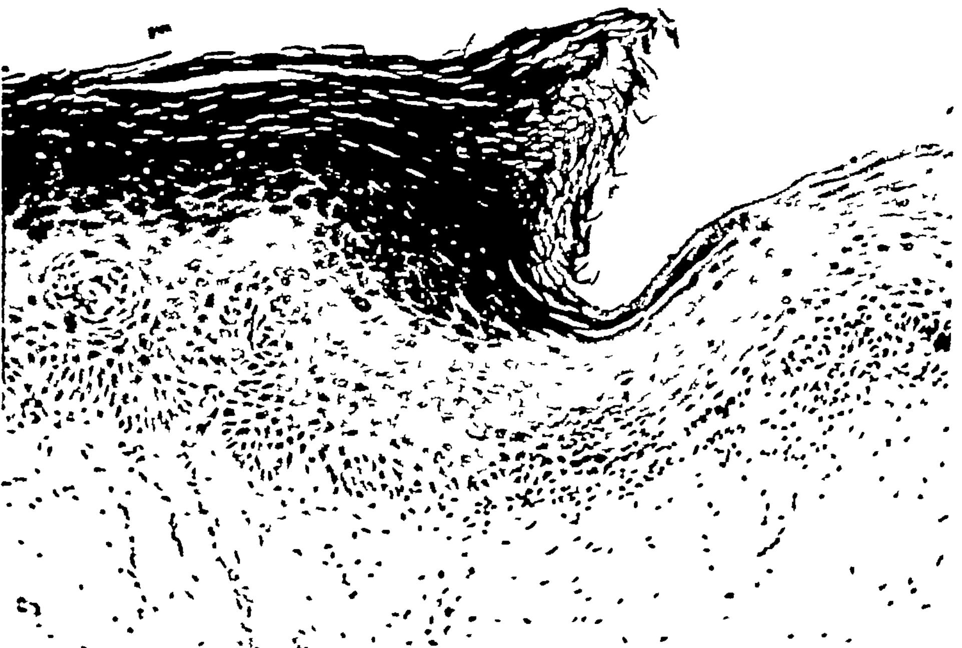


FIG 160

Oral epithelial naevus of floor of mouth Acanthosis with thick keratin and granular layer, overlying a normal corium The keratin has a basket weave appearance (*Brit J Dermat* 1956) Haematoxylin and eosin ×120



conjunction with such disorders of keratinization of the skin as generalized ichthyosis A proportion of those that occur alone are similar to the linear naevi of the skin While the floor of the mouth is a favourite site, any part of the oral mucosa may be affected often symmetrically

When the normally unkeratinized floor of the mouth is affected the increased rate of production of keratin is shown by the thick layer of keratin a thick granular layer and acanthosis (Fig 160) The keratin layer may have a basket weave appearance. There is no abnormality of the corium and the margin between the affected area and the normal unkeratinized mucosa is well defined (Fig 161)

3. REACTIONS TO EXTERNAL IRRITANTS

Ulcers. Non specific traumatic ulcers occur commonly on the oral mucosa and occasionally specific ulcers result from neoplasms or infections. The latter are described under the appropriate sections dealing with these conditions

There is a type of recurrent ulceration affecting the oral mucosa sometimes called recurrent aphthae or Mikulicz s aphthae These lesions begin as a thickening of the mucosa of the cheek lip or tongue and progress to an indolent looking punched out ulcer that may take weeks to heal Up to about four ulcers may occur at any one crop and periods of freedom from ulceration may be enjoyed for months at a time. While no explanation has so far been advanced as to their exact aetiology the sites of ulceration are to a large extent determined by trauma

Figure 162 depicts the microscopical appearance of one of these ulcers at an early stage and it is quite non specific. There is a slight undermining of the ulcerated epithelium and the dense polymorphonuclear leucocytic infiltration is localized to the breach Deeper in the corium there is a diffuse lymphocytic and plasma cell infiltration but there is nothing that sheds any light on a possible aetiological factor

Herpetic Stomatitis. Although the herpes simplex virus may affect many parts of the body it has a predilection for the epithelial cells of muco-cutaneous junctions and occasionally involves the oral mucosa. The majority of patients experience their primary infection under the age of three years, but no age is immune They are febrile and feel ill for about a week, with a pronounced submandibular lymphatic glandular involvement, sore throat, and clusters of vesicles on the vermilion border of the lips as well as distributed over the whole oral mucosa

The vesicle results from degeneration of the epithelial cells but in the mouth trauma and secondary infection soon convert the vesicle into an erosion which later heals without a scar

Confirmation of a clinical diagnosis of herpetic stomatitis is made by finding the development of specific neutralizing antibodies in the patient's serum, the isolation of the herpes virus, or by demonstrating changes in the epithelial cells affected by the virus Similar microscopical appearances may be given by epithelial cells in the vesicles of herpes zoster and varicella While histological sections of unbroken vesicles in

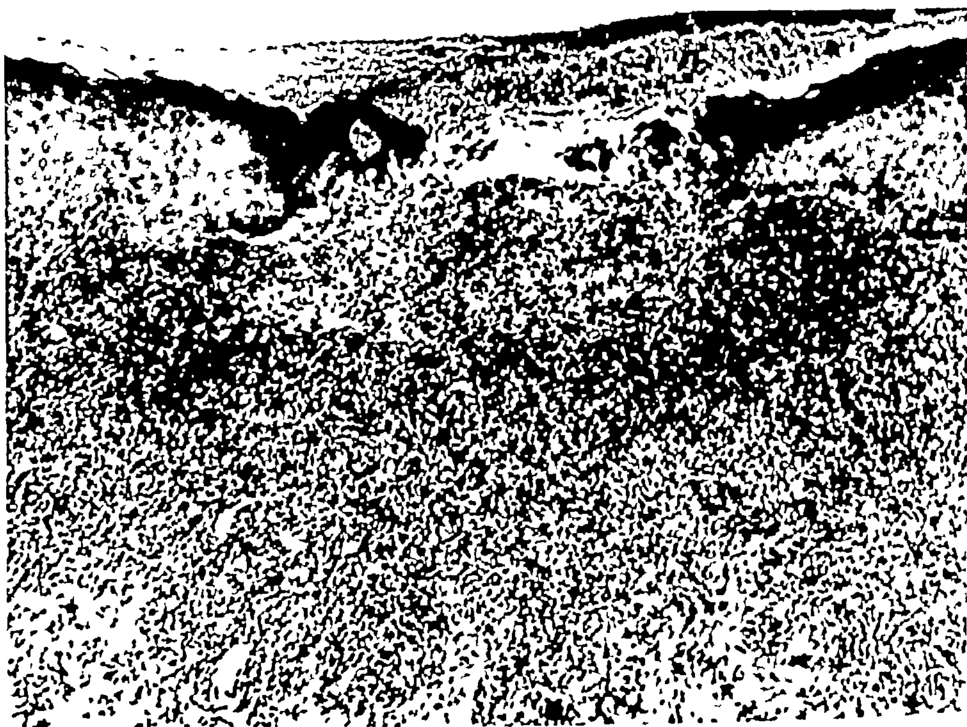


FIG 162

A recurrent aphthous ulcer There is slight undermining of the ulcerated epithelium, and the dense polymorphonuclear leucocytic infiltration is localised to the breach Haematoxylin and eosin ×70

the mouth are difficult to obtain, epithelial smears from the vesicles are a more practical diagnostic procedure In such a smear, even under low power the affected cells have a bizarre appearance, for the cells are not uniformly affected The cytoplasm is oedematous, and the large size of the cell is described as ballooning degeneration The nucleus may appear swollen in size, or there may be up to twenty or more nuclei in one cell as the result of amitotic division (Fig 163) Margination of the chromatin and small eosinophilic intranuclear bodies of Lipschutz are occasionally seen (Fig 164)

Figure 165 depicts the early formation of an intra-epidermal herpetic vesicle in the buccal mucosa The main degenerative change affecting the epithelial cells is called reticular degeneration, and only the outer



Fig 163

Epithelial smear in herpetic stomatitis. The large multinucleated epithelial cell is 80 in diameter and contains about 50 nuclei Haematoxylin and eosin ×610 (*Brit dent J* 1958)

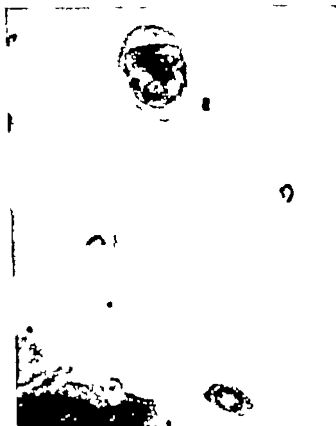


Fig. 164

Epithelial smear in herpetic stomatitis. Two cells showing eosinophilic intranuclear inclusion bodies, and margination of the chromatin Haematoxylin and eosin. ×560 (*Brit dent J* 1958)

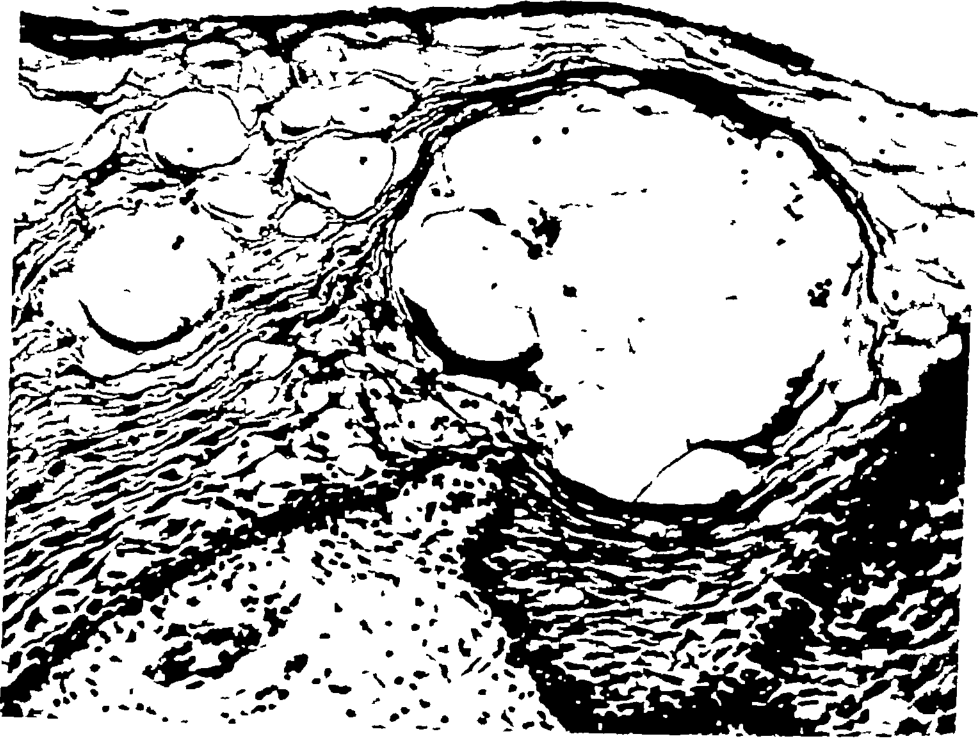


FIG 165
Reticular degeneration and formation of an intra-epidermal herpetic vesicle in the buccal mucosa Haematoxylin and eosin × 135

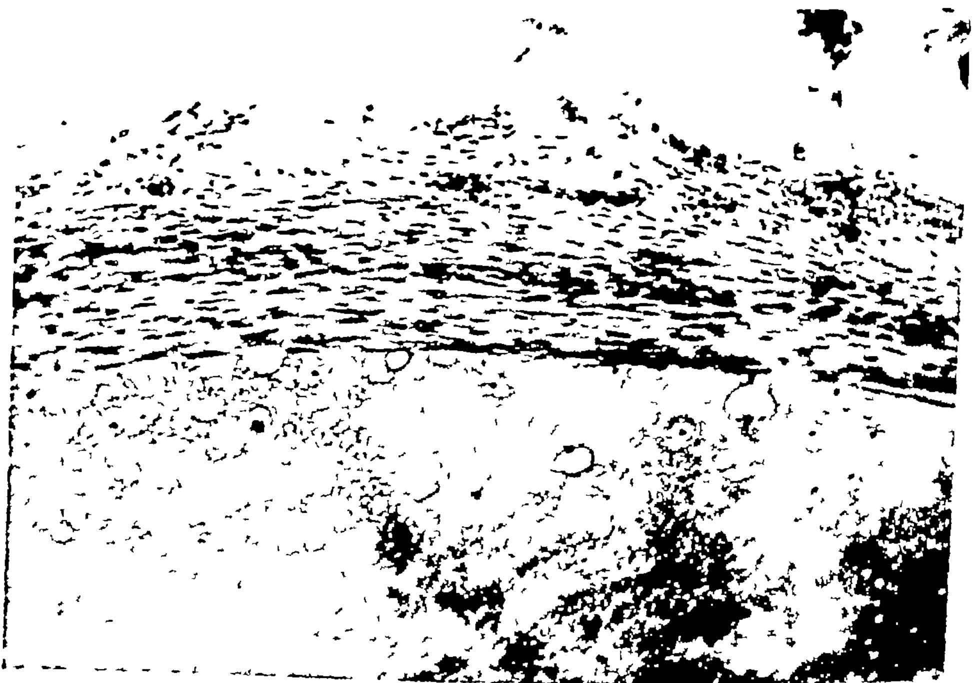


FIG 166
Mucus extravasation cyst of lower lip The mucus is contained by a condensation of fibrous tissue Haematoxylin and eosin 130

covers of the cells are retained By dissolution of adjacent cells the vesicle is formed.

Mucous Extravasation and Mucous Retention Cysts. The lower lip and cheeks are the common sites for these cysts that result from injury or inflammation of the excretory ducts of small mucous glands The duct

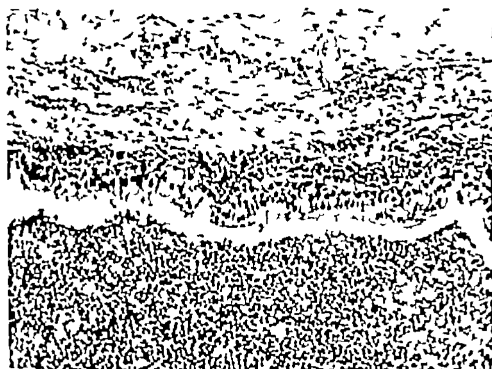


Fig. 167
Mucous retention cyst of buccal mucosa lined by columnar epithelium Haematoxylin and eosin. × 130

becomes blocked, and the proximal part dilates and then gives way under the increasing pressure of mucus. The mucus is thus extravasated into the surrounding tissues and becomes enveloped by a condensation of fibrous tissue (Fig 166)

Only very occasionally does the duct itself dilate sufficiently to form a true retention cyst. Figure 167 shows such a mucous retention cyst which is completely lined by columnar epithelium

Actinomycosis. This infection is due to *Actinomyces bovis* which is probably a normal parasite in the mouth and only becomes pathogenic when suitable paths of invasion present themselves such as the socket from which a tooth has been recently extracted

Cervico-facial actinomycosis is one of the common forms and may not cause symptoms for two to six months following an extraction Chronic abscesses form that discharge on the skin through multiple

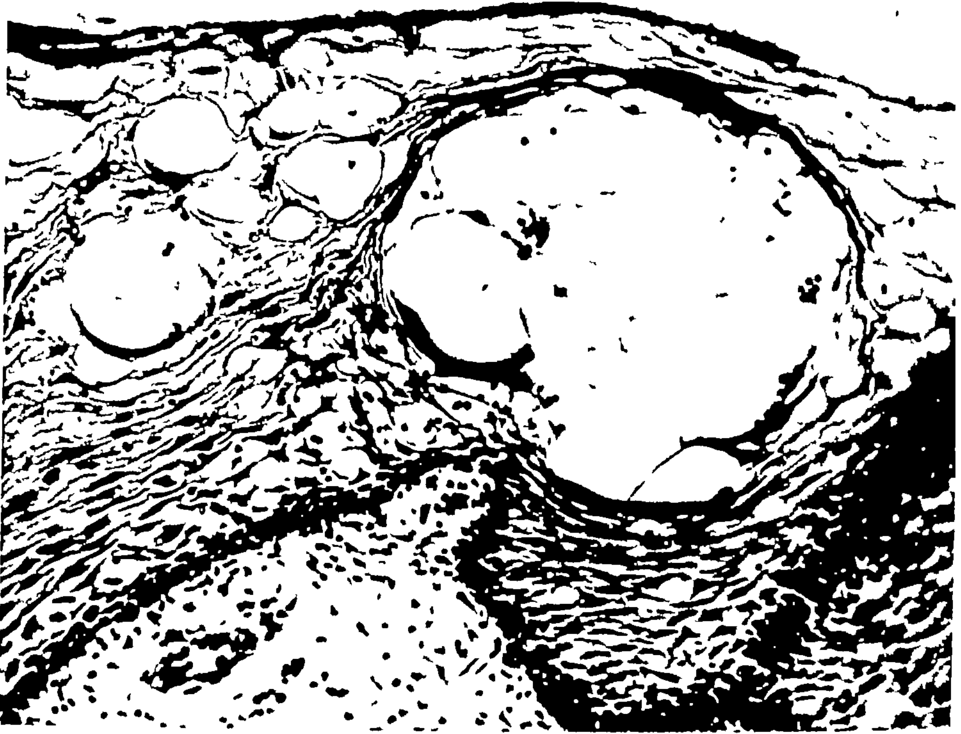


FIG 165
Reticular degeneration and formation of an intra-epidermal herpetic vesicle in the buccal mucosa Haematoxylin and eosin × 135

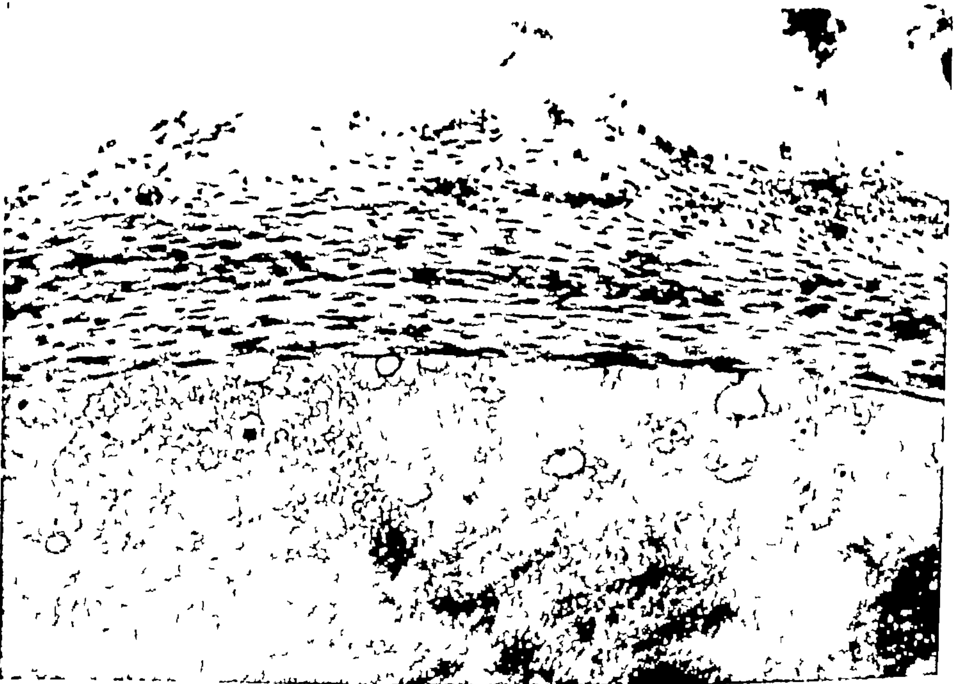


FIG 166
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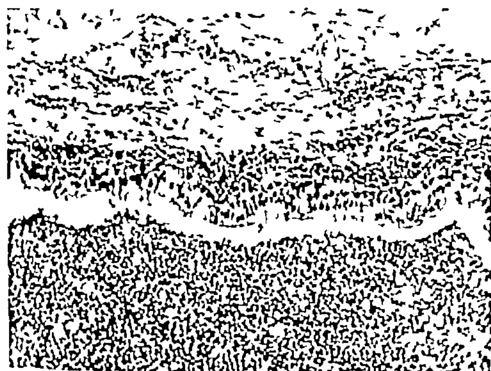


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sinuses, and healing is attended with extensive fibrosis Pus from the abscesses contains small yellow 'sulphur' granules that are visible to the naked eye, and when examined microscopically consist of a tangled mass of branching gram-positive filaments At the periphery of the granule some filaments project beyond their fellows, and end in eosino-

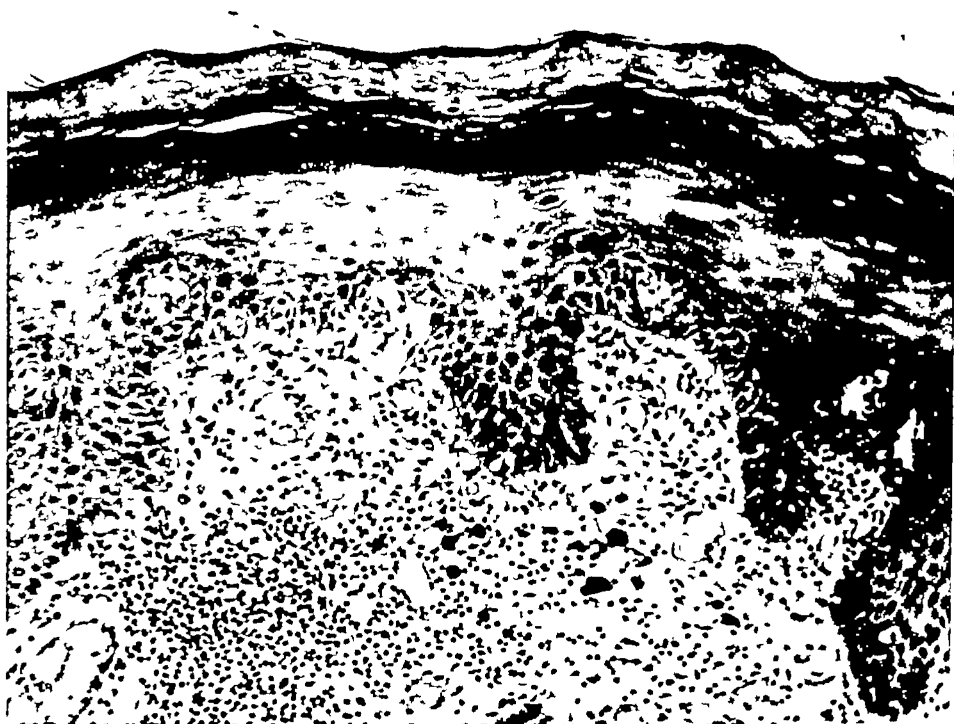


FIG 168

Leucoplakia of cheek caused by friction Parakeratosis of the epithelium overlying a corium sparingly infiltrated with lymphocytes The larger and darker spots in the middle of the corium are deposits of haemosiderin Haematoxylin and eosin × 120

philic club-like enlargements The granules are surrounded by polymorphonuclear leucocytes and foam cells

Leucoplakia. Leucoplakia is a clinical term meaning a white patch and is often used to include a number of different abnormalities of keratinization of the oral mucosa Here it will be used in the more limited sense of a reaction to external irritation Some of these lesions are precancerous, although the probability of a carcinoma supervening, except in association with syphilis, is difficult to determine Friction and smoking are external irritants that frequently precipitate this anomaly, but some individuals may have a lower threshold to keratinization than others

THE EARLY LESION The histological picture varies according to whether the mucosa affected would normally be keratinized or otherwise,

and to a less extent according to the intensity of the irritant The reaction of the non keratinized mucosa to friction or smoking may be para keratosis or keratosis, while the reaction of the keratinized mucosa to both is hyperkeratosis (Fig. 168) This is accompanied by a variable acanthosis and a very mild lymphocytic infiltration and capillary dilata



Fig. 169
Leucoplakia of cheek caused by friction Parakeratosis and a pronounced acanthosis of the epithelium overlying a corium densely infiltrated with lymphocytes and plasma cells. Haematoxylin and eosin ×100

tion in the corium (Fig 169) With the removal of the irritant, the condition may be reversible over a period of time

The presence of mucous glands in the posterior half of the hard palate modifies the reaction to smoking. The opening of the excretory duct is below the level of the adjoining keratin layer and there is a mild lymphocytic infiltration around the duct. This opening too becomes keratinized and plugs of keratin block the distal end of the duct, leading to its dilatation and the formation of a retention cyst. Thus a papule

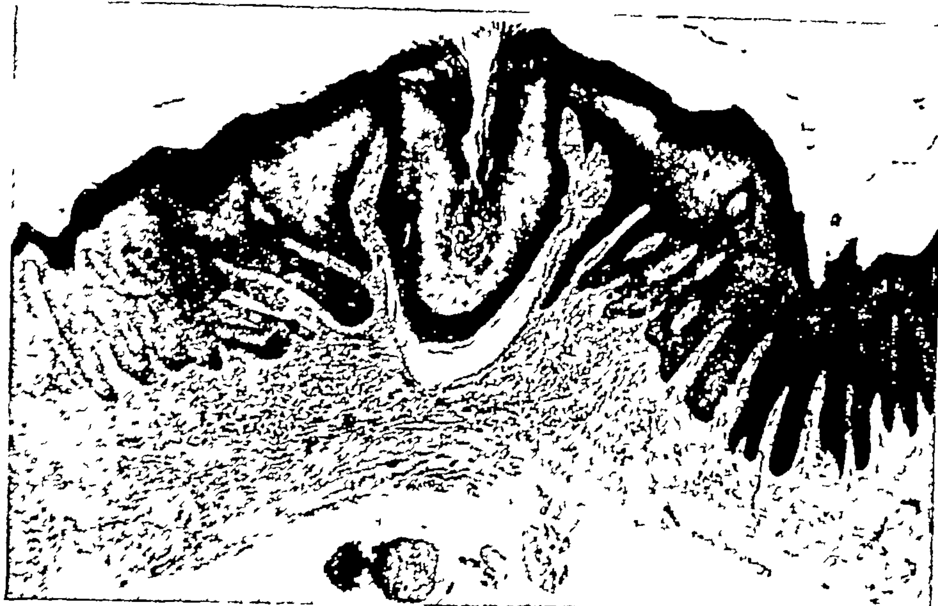


FIG 170
Leucoplakia of posterior half of hard palate caused by pipe smoking There is a plug of keratin blocking the widened orifice of a duct of a mucous gland Haematoxylin and eosin ×31 (*Brit J Dermat* 1956)

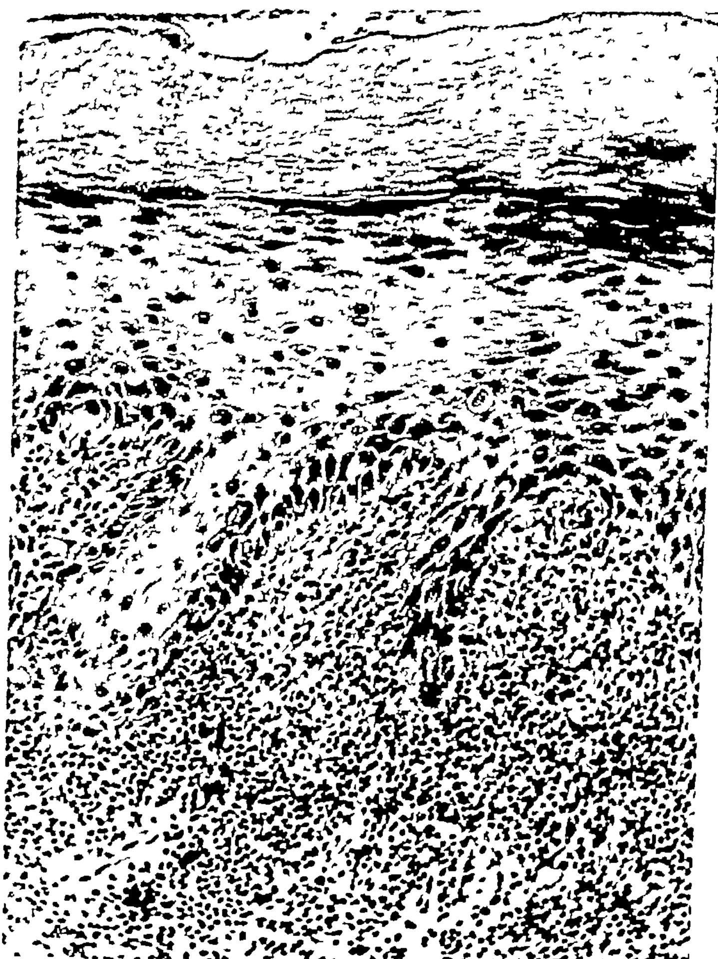


FIG 171
Leucoplakia of cheek The established lesion with a thick keratin layer and well-marked granular layer, acanthosis and a diffuse lymphocytic and plasma cell infiltration of the corium Haematoxylin and eosin ×20

results with the widened orifice of the excretory duct giving to it an umbilicated appearance (Fig. 170) A peculiar exfoliation sometimes exposing the underlying corium is observed in the adjoining keratin layer



FIG 172

Leucoplakia of tongue The late lesion with an irregularly thickened layer of keratin and some individual cell dyskeratosis and irregular downgrowth of the epithelial ridges. Haematoxylin and eosin × 100

THE ESTABLISHED LESION No matter what the cause is and whether the mucosa would normally be keratinized or otherwise, the long standing lesion will show the following cardinal features (Fig 171)

1 A thick keratin layer and a well marked granular layer

2. Acanthosis with deepening broadening and sometimes forking of the epithelial ridges.

3 Although mitotic figures are seen, there is no pleomorphism of the prickle or basal cells

4 There is often a well-defined eosinophilic condensation of the corium adjacent to the basal layer

5 There is a diffuse lymphocytic and plasma cell infiltration of the corium

THE LATE LESION In the stage of transition from hyperplasia to neoplasia (Fig 172)

1 The keratin layer varies in thickness due to desquamation

2 The acanthosis is more marked with irregular downgrowth of the epithelial ridges

3 Mitotic figures are frequent and there is individual cell dyskeratosis and clumping of nuclei

4 Many prickle cells are vacuolated and there is loss of polarity of the basal layer

5 Many of the epithelial cells are hyperchromatic

6 There is a very dense lymphocytic and plasma cell infiltration which reaches and invades the basal layer with disruption of the latter

7 There is degeneration of the elastic fibres and sometimes of the collagen fibres in the affected area

All gradations are seen between the above findings and a frank carcinoma with atypical mitotic figures, multinucleated cells, epithelial cell nests and malignant infiltration

Should there be an associated syphilitic infection, then there is also perivascular lymphocytic infiltration and endarteritis obliterans

4 REACTIONS TO INTERNAL IRRITANTS

Lichen Planus of the Oral Mucosa. Lichen planus is an inflammatory reaction of the skin and mucous membranes which on the skin is characterized by an intensely itchy eruption with the presence of papules of pink colour Oral lesions occur in conjunction with the skin lesions, but they also occur alone The oral lesion is a pearly white papule on



FIG. 173
Lichen planus of cheek A papule showing well defined keratosis and acanthosis There is dense lymphocytic infiltration with a well defined lower border Haematoxylin and eosin ×50 (*Brit dent J* 1954)

an erythematous mucosa The papules are arranged discretely in varying patterns and occur symmetrically on the cheeks tongue and less commonly on the gum Occasionally the affected mucosa is atrophic and shallow indolent erosions result from the slightest trauma

The principal histological changes in the buccal mucosa are

1 Moderately thick keratin and granular layers or parakeratosis (Fig 173)

2 Acanthosis and occasionally a saw tooth appearance to the epithelial ridges

3 Liquefaction degeneration of the basal layer (Fig 174)

4 Sub-epithelial oedema, which may separate the epithelium from the corium

5 Dense lymphocytic infiltration limited to the upper and middle layers of the corium, and having a well-defined lower margin Plasma cells are rarely present (Fig 175)

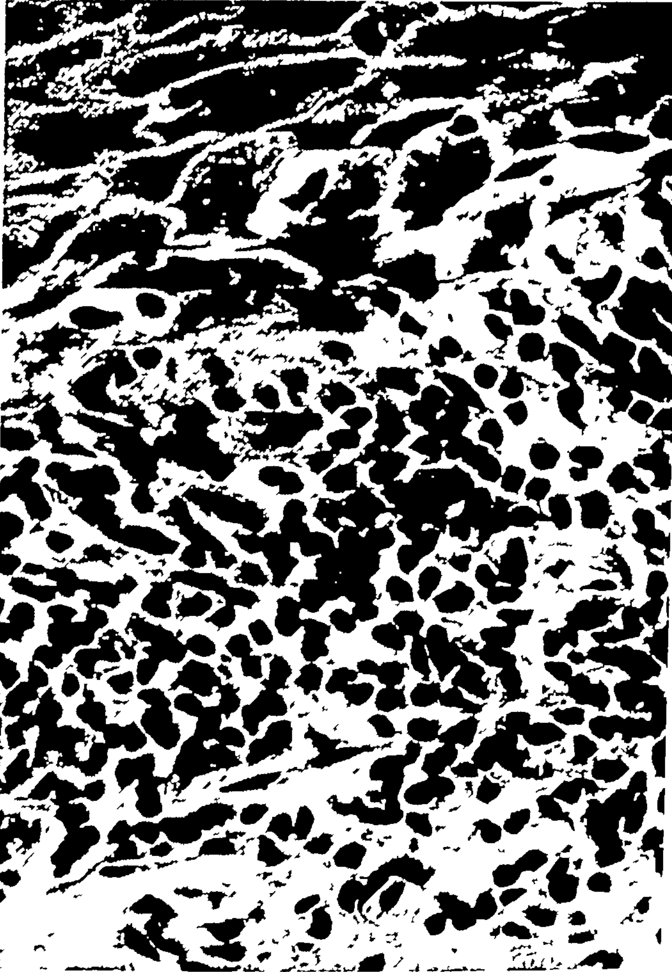


Fig 174

Lichen planus of cheek Liquefaction degeneration of the basal layer and underlying lymphocytic infiltration Haematoxylin and eosin ×535 (*Brit dent J* 1954)

6 In the atrophic form, the keratosis or parakeratosis is associated with atrophy of the epithelium The changes in the dermis are similar to the foregoing (Fig 176)

Great difficulty may be experienced in distinguishing the microscopical appearances of lichen planus from those of leucoplakia, but in general it may be said that neither the keratosis nor the lymphocytic



Fig. 175

Lichen planus of cheek. Parakeratotic epithelium overlying a dense lymphocytic infiltration limited to the upper and middle layer of the dermis. Haematoxylin and eosin. × 50



Fig. 176

Atrophic lichen planus of cheek. Keratosis and atrophy of all the other layers of the epithelium, and a band-like distribution of the lymphocytic infiltration. Haematoxylin and eosin × 130 (*Brit dent J* 1954)

infiltration in leucoplakia is so localized as in lichen planus The infiltration in leucoplakia is generally less dense and plasma cells, if not plentiful, may be found It is only in leucoplakia that transitional changes between hyperplasia and neoplasia are seen in the epithelium

5. NEOPLASMS

BENIGN EPITHELIAL NEOPLASMS

Squamous Papilloma. The papilloma is a benign neoplasm of epithelium Unfortunately the term ' papilloma ' only describes its papillary or cauliflower macroscopic appearance, which a carcinoma may mimic It may arise on the uvula, palate, tongue, cheeks, lips and less commonly from the gum It may be soft and red, or firm and white according to the degree of keratinization The heavily keratinized papilloma may be associated with a patch of leucoplakia While the papilloma on the oral mucosa has not the bad prognosis associated with similar lesions in the intestines and bladder, it is perhaps wise to consider it as a potentially precancerous lesion, especially when associated with leucoplakia

Histological examination reveals finger-like processes of hyperplastic stratified squamous epithelium which are supported by thin cores of fibrous connective tissue The epithelium may or may not be keratinized, mitotic figures are sparse and in any one field it may lack neoplastic features (Figs 177, 178)

Pleomorphic Adenoma of the Salivary Glands. (' *Mixed Salivary Tumour* ') This is a salivary epithelial tumour that in behaviour varies between being benign and malignant, and all transitions are seen between the slowly growing, highly differentiated adenoma and the more rapidly growing, poorly differentiated adenocarcinoma These tumours arise not only from the major salivary glands, but also from the minor glands in the posterior half of the palate, cheeks, lips and very rarely from the gum The majority derived from the minor glands present as slowly growing, soft, sessile tumours Willis (1948) prefers the term pleomorphic adenoma to mixed tumour, for all the evidence supports their being epithelial alone in origin ' Pleomorphism is a striking feature of this group of tumours '

There is a tendency for them to recur after enucleation, for although macroscopically they may appear encapsulated, microscopically they are not, and because of their slow growth, too conservative operations are sometimes performed Sometimes a similar neoplastic change may arise in adjacent glandular tissue giving rise to satellite tumours



FIG. 177

Low power photomicrograph of a well keratinized papilloma from the gum showing the finger like processes of hyperplastic stratified squamous epithelium. *Haematoxylin and eosin* ×40



FIG. 178

Well keratinized papilloma from the palate. The finger like process of epithelium is supported by a thin core of fibrous connective tissue. Haematoxylin and eosin ×60



FIG 179

Pleomorphic adenoma arising from mucous glands in the hard palate Normal mucous acini are to the left, while to the right epithelial cords spread in a plexiform manner, with here and there an abortive ductular pattern Haematoxylin and eosin ×120

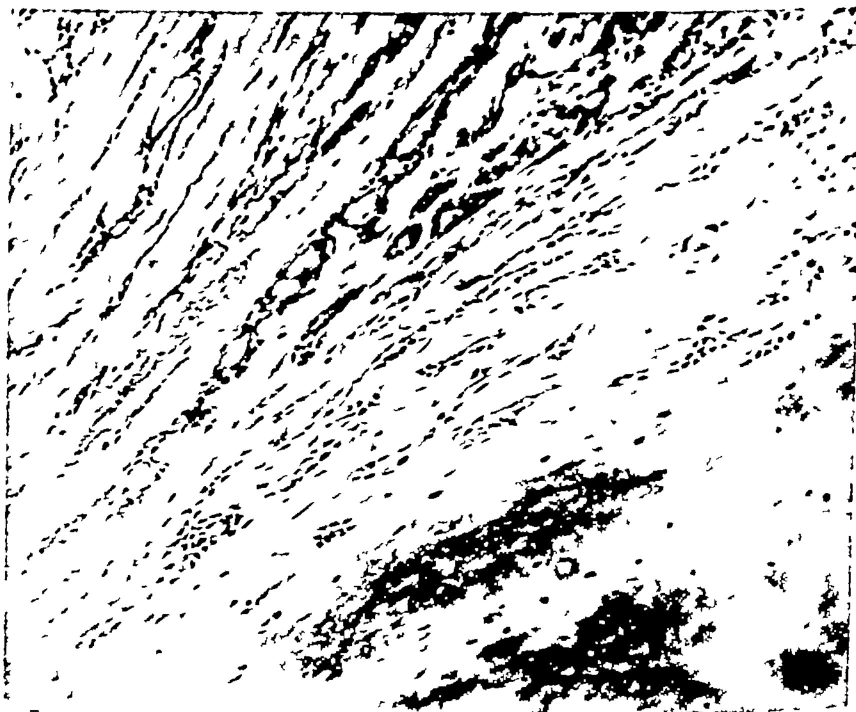


FIG 180

Pleomorphic adenoma arising from mucous glands in the hard palate 'Cartilage-like' areas, where the epithelial cells have become widely separated from each other in the mucinous matrix Haematoxylin and eosin ×120

The following microscopical appearances may be observed

1 Normal glandular tissue in the transitional area between the tumour and the parent gland (Fig 179)

2. Atypical glandular tissue partly acinar and partly ductular that may form convoluted and plexiform patterns. Solid epithelial cords sprout from the border of the acini

3 Cartilage like areas where the epithelial cells have become widely separated from each other in a mucinous matrix (Fig. 180) This mucinous matrix is not a mucoid change in the connective tissue stroma but is the result of the mucus of the epithelial parenchyma mixing with the stroma In other parts the collagen fibres undergo hyaline change

4 The condensation of fibrous tissue forming the capsule may show infiltration by epithelial cells

MALIGNANT EPITHELIAL NEOPLASMS

Squamous-cell Carcinoma The squamous-cell carcinoma is the most common form of malignant neoplasm affecting the oral mucosa The basal and the prickle cells are both involved in the tumour All gradations are seen between the well differentiated and keratinized lesions and those that are undifferentiated and anaplastic. The well differentiated squamous-celled carcinoma that occurs in skin does not often occur in the mouth It is characterized by infiltration of the corium by irregular branching trabeculae of well differentiated prickle cells The prickle cells are large with vesicular nuclei and their prickles are readily visible Mitotic figures are plentiful but except in the deepest part of the growth there is a limiting layer of basal cubical cells. Other trabeculae are arranged as cell rests, with the cells arranged in whorls of keratin The centre of the whorls may be completely keratinized or there may be a single degenerate cell or just débris while at the outer margin there is a limiting layer of basal cells There is often a dense lymphocytic and plasma cell infiltration around the epithelial trabeculae together with a sprinkling of eosinophils (Fig 181)

The majority of the squamous-celled carcinomata of the oral mucosa are less differentiated. The cell rests are sparse and less well keratinized and the prickle cells are much more pleomorphic. The latter vary greatly in size and shape and many of their nuclei are hyperchromatic. Their prickles are no longer easily identifiable Mitotic figures are abundant and many of them are atypical with many bizarre forms (Fig 182)

In the very undifferentiated carcinoma the epithelial cells are scarcely recognizable as prickle cells and are hard to distinguish from the stroma Instead of cell nests, individual cells undergo keratinization, a feature sometimes called malignant dyskeratosis.



FIG 181
Well-differentiated squamous-celled carcinoma with many keratinized epithelial whorls, and branching trabeculae of well-differentiated prickle cells There is a dense lymphocytic infiltration of the adjacent stroam Haematoxylin and eosin × 60

Adeno-cystic Carcinoma. This is an important variant of the adeno-carcinoma and may arise from any salivary tissue, and particularly from the mucous glands of the palate It is highly invasive and may metastasize early It is a very cellular tumour with little mucous or stromal participation Some fields show trabeculae of rather undifferentiated epithelial cells with a cribriform pattern These tumour cells tend to have an acinar arrangement with short cubical cells surrounding spaces containing an eosinophilic structureless material (Fig. 183) In other

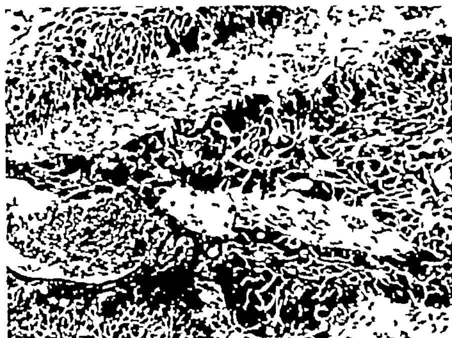


FIG. 182
Poorly differentiated squamous-celled carcinoma of the gum with many bizarre shaped prickle cells and hyperchromatic nuclei Haematoxylin and eosin × 120

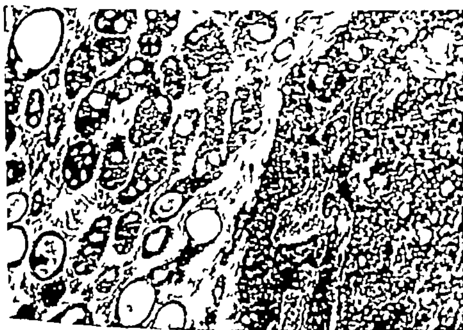


FIG 183
Adenocystic carcinoma of palate with trabeculae of undifferentiated epithelial cells tending to form acini to give a cribriform pattern Haematoxylin and eosin. × 110 (*Brit dent J* 1955)

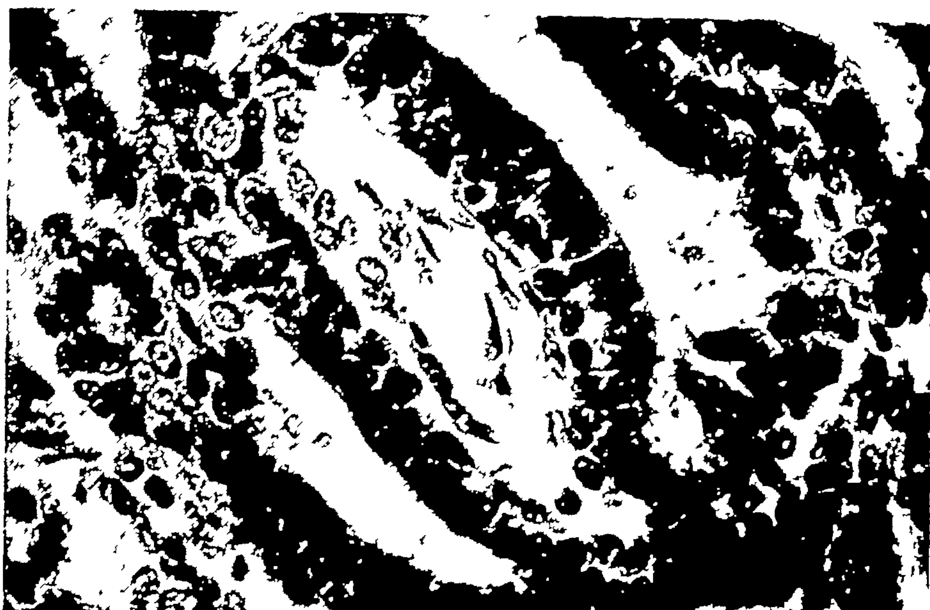


Fig 184
Adenocystic carcinoma with a well-defined arrangement of ducts, the ducts being lined with a layer of tall columnar cells resting on a basal layer Haematoxylin and eosin ×470 (*Brit dent J* 1955)



Fig 185
Prit f the hard palate,
poly s abut on the prick
overl eeper in the cort
nted and fusifor

fields there may be a pattern of well differentiated ducts the ducts being lined with a layer of tall columnar cells resting on a basal layer (Fig 184)

Both basal-celled carcinomata and adamantinomata may present a pseudo-glandular appearance but neither would give the ducts as seen in this tumour

Malignant Melanoma Although these neoplasms are fortunately very rare, they deserve special consideration for not only may secondaries form in the oral mucosa but the hard palate would appear to be a site of predilection for the primary growth They are the most malignant of all the neoplasms occurring in the mouth

Figure 185 depicts a primary growth on the hard palate and the *naevus cells* show great pleomorphism The basal layer of the overlying epithelium is missing and unpigmented polyhedral naevus cells abut on the prickle layer Deeper in the corium the naevus cells are pigmented fusiform and closely packed together

It is not known whether the malignant melanoma arises from a pre-existing developmental mole on the mucosa but as shown in the pigmentation of Addison s disease the oral mucosa is capable of producing melanin

BENIGN CONNECTIVE TISSUE NEOPLASMS

The Fibroma is a benign connective tissue tumour the cell type of which is a fibroblast This slowly growing tumour is rare compared with the common fibro-epithelial polyp of inflammatory origin

The microscopical appearance is that of fibrous tissue covered with a thin layer of normal epithelium (Figs 186 187) The fibroblasts and collagen fibres may have a distinct whorled pattern The more cellular lesions show plump spindle fibroblasts and few collagen fibres, while others may show dense collagen bundles with a hyaline appearance and only a few fibroblasts Woven bone formation is an occasional finding (Fig. 188)

The Neurofibroma is a tumour that arises from all the elements of a peripheral nerve It is benign and generally solitary although it may be multiple as in neurofibromatosis It is not a well-defined tumour like the schwannoma but mingles with the surrounding normal tissue The main mass of the tumour consists of fibrous or mucoid tissue among which nerve fibres may be seen The fibrous tissue is arranged in a whorled pattern, but it lacks the regimentation of a schwannoma (Fig. 189)

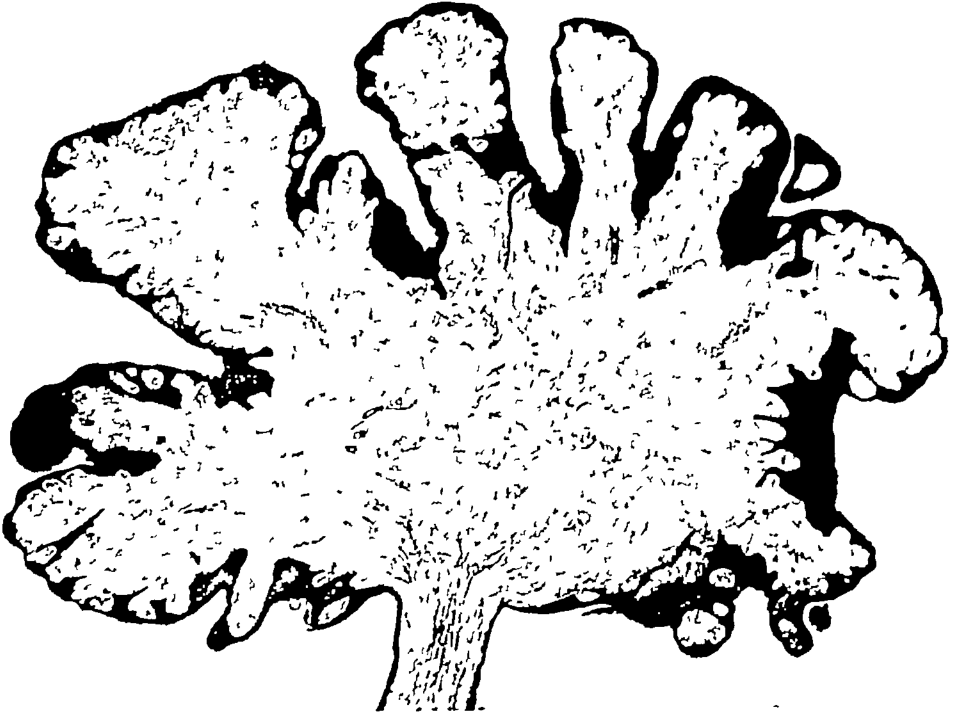


FIG 186
A low power photomicrograph of a young fibroma from the gum Haematoxylin and eosin ×21



FIG 187
A higher magnification of part of the fibroma illustrated in Figure 186 Fibroblasts and collagen fibres covered by a thin layer of normal epithelium Haematoxylin and eosin ×120



FIG. 188
Fibroma of gum with woven bone formation. Haematoxylin and eosin × 11



FIG. 189
Neurofibroma of mandibular nerve with whorled pattern of fibroblasts adjacent to areas of mucoid tissue Haematoxylin and eosin. × 120

Von Recklinghausen's neurofibromatosis is a developmental disorder characterized by patches of cutaneous pigmentation and the development of multiple neurofibromata Sarcoma may supervene

The schwannoma is a nerve sheath tumour that arises from the neurilemmal cells of Schwann It is a benign solitary tumour and apart



FIG 190
Fibro-lipoma of cheek Vacuolated fat cells (from which the fat has been lost in the preparation of the paraffin section) supported by fibrous tissue Haematoxylin and eosin × 30

from the acoustic nerve, the cranial nerves are rarely the site of this lesion

The Lipoma and Fibro-lipoma. The lipoma is a benign connective tissue tumour derived from mature fat cells When there is also a large fibrous element it is called a fibro-lipoma (Fig 190) These tumours arise most commonly in the submucosa of the cheeks or lips

The Haemangioma is a vascular tumour commonly found in the skin and less commonly in the tongue and buccal mucosa Very few

haemangiomata are neoplasms for some are present at birth or are noticed early in life and should be considered as developmental anomalies, growing in proportion with the rest of the body Haemangiomata are often multiple and many syndromes are described one of the most



FIG 191
Haemangioma of gum The capillary spaces in the upper half of the photomicrograph are lined only by endothelial cells, while in the lower half there are solid trabeculae of endothelial cells that are imperfectly canalised Haematoxylin and eosin. × 120

important being hereditary multiple telangiectasia of skin and mucous membranes

A false clinical appearance of neoplastic enlargement may be given by accidents such as thrombosis and oedema. In fact many small lesions in the oral mucosa are only brought to light when they are injured and the subsequent thrombosis gives rise to a firm palpable nodule Other haemangiomata are just vascular reparative tissue or hi vascular connective tissue

Capillary, cavernous and plexiform types occur in various combinations Solid trabeculae of endothelial cells that are imperfectly canalized may give a neoplastic appearance (Fig. 191).



FIG 192
Lymphangioma of gum, with foci of lymphocytes in the supporting tissue between the spaces lined only with endothelial cells and containing lymph Haematoxylin and eosin × 120

The Lymphangioma is a tumour of lymph vessels and like the haemangioma is generally a developmental anomaly. It is a very uncommon lesion in the mouth Figure 192 illustrates the lymphangioma of the gum There are spaces lined only with endothelial cells and containing lymph and lymphocytes There are also foci of lymphocytes in the supporting tissue between the vessels

MALIGNANT CONNECTIVE TISSUE NEOPLASMS

Sarcoma. A sarcoma is a malignant tumour arising from any mesodermal tissue, and the prefix fibro-, lipo-, osteo-, etc., pertains to the parent cell. Sarcomata are fortunately rare in the mouth, for the prognosis is extremely poor, metastases to the lungs through the blood stream being frequent

Since the undifferentiated mesenchyme cell is [illegible] t of the more differentiated types of mesenchyme such as f [illegible] d bone, it

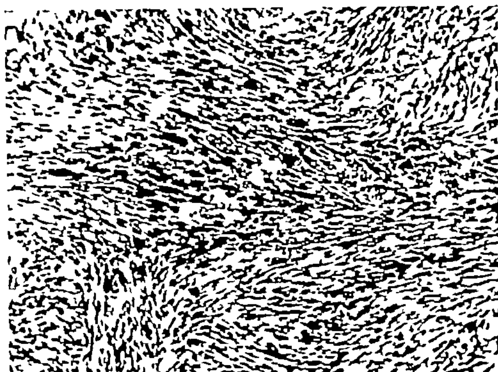


FIG. 193
Fibro-sarcoma of mandible with great pleomorphism amongst the fibroblasts. Haematoxylin and eosin ×120 (From a specimen kindly lent by Professor R B Lucas.)

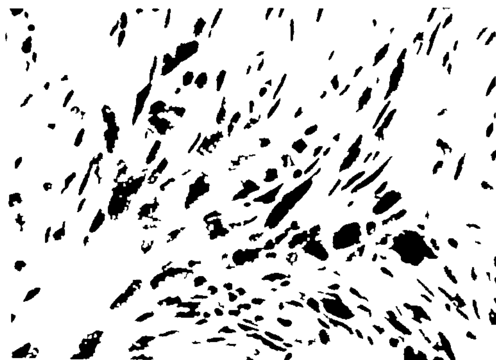


FIG. 194
Fibro-sarcoma of mandible with many irregular multinucleated fibroblasts. Haematoxylin and eosin ×430 (From a specimen kindly lent by Professor R. B Lucas.)

may not always be possible from histological examination alone to identify the origin of a given tumour While there is a tendency for the osteo-sarcoma to form bone, the fibro-sarcoma may also form bone, and in no other group of tumours is metaplasia so common

A typical fibro-sarcoma will show pleomorphism among the fibroblasts, with many mitotic figures and large irregular multinucleated cells (Figs *193*, *194*)

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IV

THE MANDIBLE AND THE MAXILLA

1 DEVELOPMENTAL ANOMALIES

Fibrous Dysplasia of Bone Fibrous dysplasia is characterized by fibro-osseous lesions that have a tendency to stabilization when skeletal growth has ended and is not associated with any disturbance of calcium metabolism It is probably a developmental anomaly of the bone forming mesenchyme Many bones or only a small part of one bone may be affected The more florid cases involving many bones may be accompanied by cutaneous pigmentation and endocrine disorders While the jaws may be involved in the polyostotic cases they are more often involved alone, and the term facial fibrous dysplasia has been coined by Thoma (1954) to describe this group

The *microscopical appearances of fibrous dysplasia do not suggest* any neoplastic quality and the most striking feature is the regular manner in which the trabeculae of woven bone are formed and then *completely resorbed or partially remodelled (Fig 195) As these changes* become less rapid lamellar bone is formed and after complete arrest the trabeculae have a most inactive appearance

None of the original bone that has been replaced by fibrous tissue remains and the latter does not have any well marked whorled pattern The thin trabeculae of woven bone are laid down about equidistant from one another and in any one field they appear to reach the same stage of maturity (Fig 196)

Developmental Cysts *of the Jaws that are not Odontogenic in Origin.* These cysts arise from enclaved epithelium at the lines of fusion of embryonic processes and from the remnants of vestigial structures They are thus *constant in site and unrelated to the position or vitality of* neighbouring teeth. Their importance is in connexion with differential diagnosis so that teeth are not needlessly sacrificed in their treatment

THE NASO-PALATINE CYST is an epithelium lined developmental cyst arising from vestiges of the naso-palatine ducts It is commonly called the incisive canal cyst for it forms in that canal

THE GLOBULO-MAXILLARY CYST is a developmental fissural cyst arising from enclaved epithelium following the fusion of the globular and the maxillary processes, and thus is found *between the maxillary* lateral and canine teeth

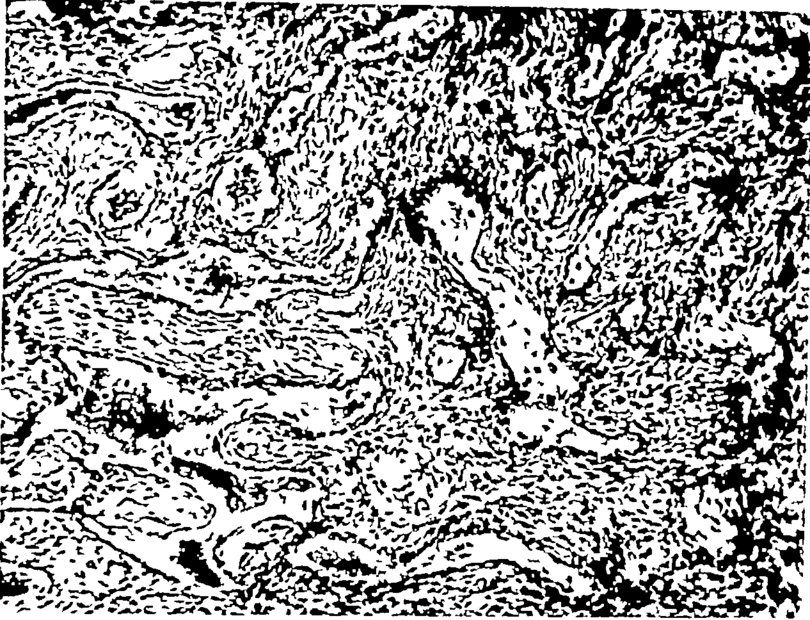


Fig 195

Fibrous dysplasia of R maxilla Fibrous tissue and thin trabeculae of osteoid and woven bone Haematoxylin and eosin × 100 (*Brit dent J* 1957)

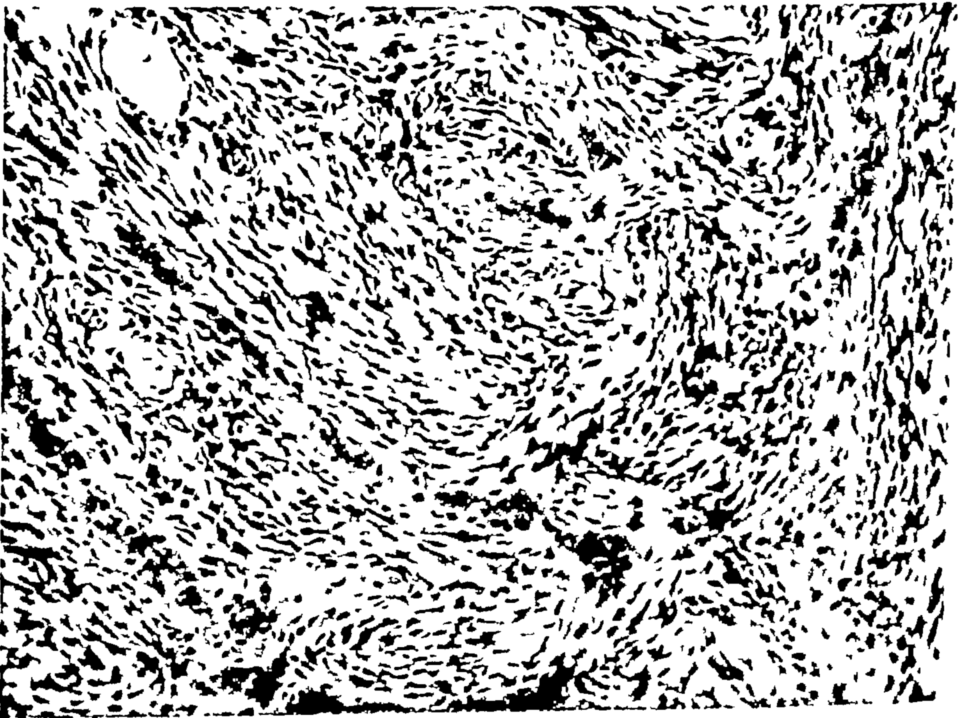


Fig 196

Fibrous dysplasia of mandible Thin trabeculae of woven bone in fibrous tissue Haematoxylin and eosin × 130

THE NASO-LABIAL CYST is derived from the same epithelial remnants as the globulo-maxillary cyst but is situated entirely in the soft tissues anterior to the bone.



FIG. 197
Naso-palatine cyst lined by pseudo-stratified columnar epithelium. Haematoxylin and eosin ×280 (From a specimen kindly lent by Professor R B Lucas.)

Since the parent cells from which these cysts arise may be respiratory or oral in type according to their proximity to the nasal or oral cavity the cysts will be partly or wholly lined by either pseudo-stratified columnar or stratified squamous epithelium (Fig. 197) The lining rarely shows evidence of infection.

2. INFLAMMATION

Osteomyelitis of the Jaws. Since with the extraction of every tooth the bone of the jaws is exposed to the oral fluids it is surprising that osteomyelitis of the jaws is not more common Should a tooth be extracted in the presence of an acute pericoronal infection then osteomyelitis may supervene. The organisms often implicated include the *Staphylococcus aureus* and *albus* and the *Streptococcus haemolyticus* As in other bacterial infections the outcome depends upon the virulence and number of the bacteria and the resistance of the host. When the bone is thin and has a generous blood supply as in the maxilla, osteomyelitis



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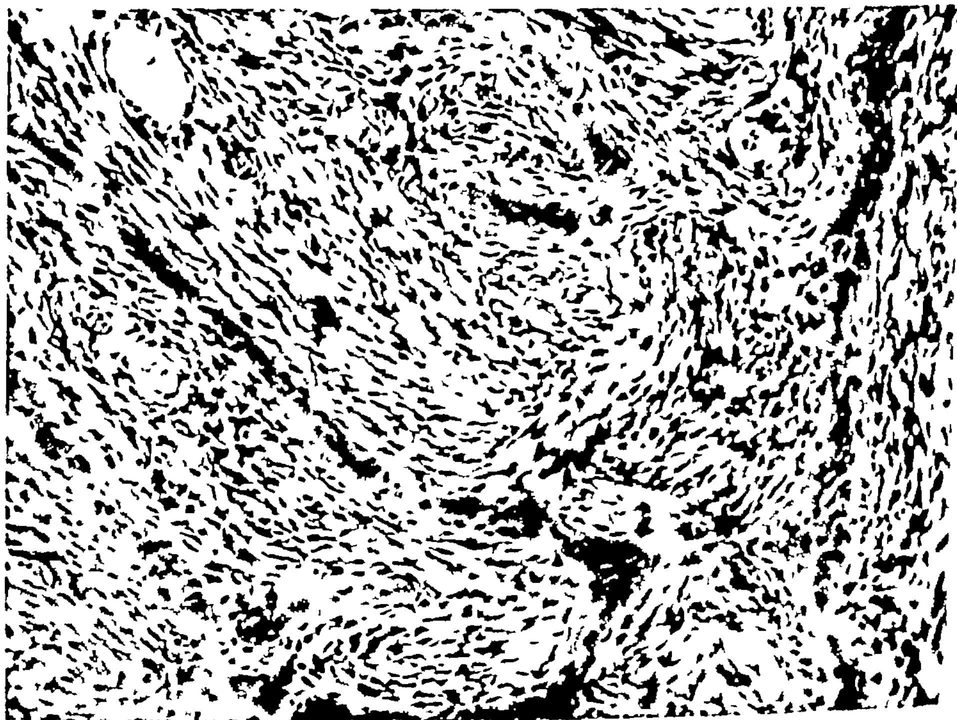


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is uncommon and quickly localized In the mandible with its thick trabeculae or cortical bone chiefly dependent upon the mandibular artery for its blood supply, osteomyelitis is more frequent and diffuse, and large sequestra may result

The Acute Infection and Necrosis of Bone From the portal of entry, be it a socket, a fracture line or a blood vessel, the organisms proliferate in the marrow spaces giving rise to dilatation of the capillaries, oedema and polymorphonuclear infiltration In such a confined space the reparative reaction is limited and thrombosis of neighbouring vessels quickly follows with death of the marrow cells and the formation of pus The bone surrounded by the pus dies, and the lacunae appear empty Since osteoclasts and osteoblasts are living cells, no resorption or apposition can affect the necrosed bone while it is surrounded by pus The raised intra-medullary pressure forces the pus along paths of least resistance such as through other marrow spaces to the periosteum The periodontal membrane is another escape route taken by pus from the centre of the jaw to its surface

Sequestration of Necrotic Bone and the Formation of an Involucrum The necrotic bone is separated from the neighbouring vital bone to become a sequestrum, by osteoclastic action at the expense of the living bone Granulation tissue proliferates in the area formerly occupied by pus, and osteoclasts will be found on surfaces of the sequestrum undergoing resorption In like manner, granulation tissue proliferates in the space beneath the periosteum that was lifted from the surface of the bone by pus in the acute phase, and in this granulation tissue an involucrum is formed of coarse-fibred woven bone

While large sequestra are exfoliated with or without surgical intervention, very small ones may be completely resorbed or have woven bone laid down on their surfaces As the sequestra are being exfoliated slowly through sinuses in the gum, they may become partially enveloped by squamous epithelium Sequestra exposed to mouth fluids may also undergo proteolysis and decalcification in a manner similar to caries of cementum The organisms invade the bone in planes parallel to the line of the fibres, and along the canaliculi In this manner the bone is broken down by oral acidinogenic and proteolytic bacteria acting in a moist environment.

The Final Reparative Phase Eventually much of the woven bone that forms the involucrum and fills the cavities formerly occupied by sequestra is remodelled and replaced by lamellar bone, which finally assumes an architecture approximately identical with that of the neighbouring unaffected bone Fibrous tissue filling the marrow spaces may remain as the only permanent scar Most of the subperiosteal bone is

resorbed although the jaw may be left a little thicker than it was originally

3 REPAIR AND REGENERATION

Healing of Fractures. IMMEDIATE RESULTS. In complete fractures the periosteum is torn and the adjacent soft tissues are damaged The neighbouring marrow degenerates and the bone around the fracture lines becomes necrotic. Haemorrhage occurs between the fractured ends resulting in a haematoma The haematoma organizes and macrophages and polymorphonuclear leucocytes remove necrotic tissue The fibroblasts lay down collagen fibres mainly parallel to the long axis of the bone, surrounding the fractured ends filling the space between the fragments and sealing the marrow spaces If the fracture is simple and uncompli cated by infection the signs of inflammation disappear within a few days and regenerative changes follow

REGENERATION *The Provisional Callus* Callus is divisible according to its origin and site into

1 outer (*a*) periosteal beneath the periosteum (*b*) parosteal within adipose and fibrous tissue
2. inner endosteal or medullary callus in the medulla
3 intermediate or osteal between the fractured osseous surfaces

The Outer Callus Both beneath the periosteum and in the adjacent connective tissue osteoid tissue forms, resembling that seen in foetal periosteal growth Calcification of the osteoid tissue takes place rapidly resulting in coarse fibred woven bone

Cartilage may be found in the callus, and is most abundant where there is mobility of the fractured ends This cartilage is later replaced by bone by endochondral ossification

The Inner Callus is less important it protrudes into the marrow spaces and is maximal at the fracture line

The Intermediate Callus is the last to form and follows after resorption of the fractured ends, while the space is filled with fibrous tissue. Thus the fracture line widens before it is united by the intermediate callus.

The provisional callus then, is composed of coarse fibred woven bone, some lamellar bone with possibly some cartilage in addition forming a spongy spindle-shaped mass considerably in excess of what is needed for the union of the two ends The time taken for this callus to form varies with the age and health of the patient and the site in the bone affected. The provisional callus is so radiolu[illegible]
not show on radiographs

Definitive Bony Callus Later the provisional callus is converted to dense lamellar bone as a result of lacunar resorption and the apposition of fresh lamellae. The final stage in callus formation is not reached until the bone has been in function for some time, and the lamellar bone has had its architecture orientated according to the stresses and strains it has to bear

It is difficult to generalize about approximate times that these stages take but Weinmann and Sicher (1955) suggest that in uncomplicated cases the haematoma coagulates six to eight hours after the accident, and organization may be completed in one to two weeks, after which the provisional callus forms Final remodelling of the definitive callus may take months or years

The Healing of Extraction Wounds. The pattern of healing of an extraction wound is similar to that of the healing of a fractured bone Nevertheless, because the wound is in direct contact with the mouth, there is a greater chance that the blood clot will become infected and break down, thereby delaying the healing process.

The blood filling the socket clots and protects the bony surface of the socket Organization of the clot proceeds most rapidly in the upper third of the socket due to the greater vascularity of the adjacent submucosa The exposed part of the clot shows a polymorphonuclear leucocytic infiltration in the early stages, but during the second week epithelium proliferates from the margins of the socket across the organizing blood clot At the same time coarse-fibred woven bone begins to form in the fibrous tissue filling the socket and osteoclastic resorption of the lamina dura occurs In later weeks after the woven bone has filled the socket it undergoes resorption and is replaced by lamellar bone The lamina dura is completely resorbed The final pattern results in some loss of bone in the area, and a compact plate is formed on the surface giving a new margin to the alveolar crest The final picture may be modified by the movement of adjacent teeth

If following the extraction the blood clot becomes infected resulting in a dry socket, then healing cannot begin until the infection has been overcome enough to allow granulation tissues to proliferate from the apical area This granulation tissue then provides the scaffolding for woven bone formation, and healing proceeds but at a slower rate than for the uninfected socket

The Central Giant-cell Tumour and the Giant-cell Reparative Granuloma. It is extremely difficult to ascertain by microscopical examination alone whether a given central giant-cell lesion is a neoplasm or a reparative granuloma, or even a 'brown tumour' of hyperparathyroidism and all three lesions may occur in the jaws

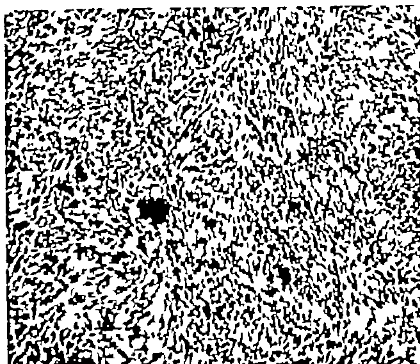


FIG 198
Central giant-cell tumour of the maxilla with the giant cells arranged focally in sheets of cellular fibrous tissue Haematoxylin and eosin. ×100

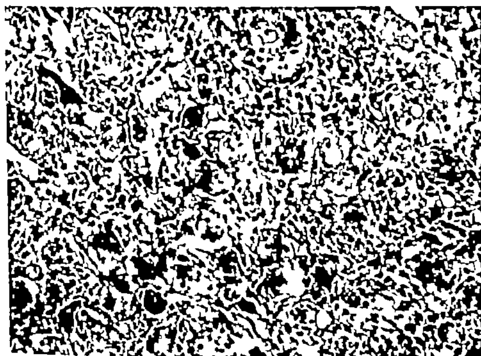


FIG. 199
Central giant-cell tumour of maxilla with the giant cells arranged in a syncytial manner and polygonal stromal cells Haematoxylin and eosin. ×145

Jaffe (1953) believes that the majority of the central giant-cell lesions of the jaws are reparative reactions, with the giant cell related to areas of haemorrhage (Fig 198) Trabeculae of woven bone may be formed Their clinically benign behaviour and their successful treatment with

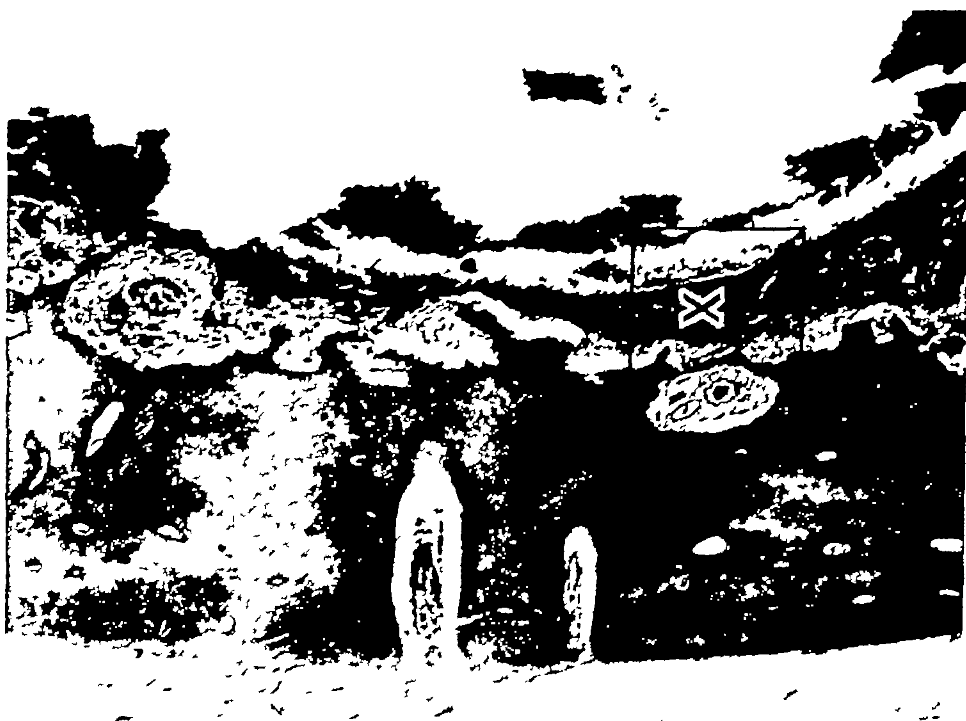


FIG 200
Traumatic bone cyst of mandible The outer plate of bone is undergoing resorption and is separated from the cyst cavity by a layer of fibrous tissue Haematoxylin and eosin × 31

curettage contrast with the truly aggressive giant-cell tumours of the ends of the long bones The age incidence of ten to fifteen years for the jaws is below that for the long bones

Nevertheless there are clinically aggressive giant-cell tumours of the jaws, which recur after curettage, and could be regarded as neoplasms In these, the osteoclasts are perhaps more plump and arranged in a syncytial manner rather than focally, while the stromal cells are polygonal or spindle shaped, with little collagen formation and no bone (Fig 199)

In many central giant-cell lesions, the possibility that it may be a ' brown tumour ' occurring in hyperparathyroidism should be considered This brown tumour is in fact a reparative reaction Valuable confirmatory evidence is a raised serum calcium level or radiographic changes in other bones

The Solitary or Traumatic Bone Cyst. The solitary bone cyst is an *uncommon* [illegible] *found in the mandible, which on so many occasions has*

followed an injury that it is often called a traumatic bone cyst It is found in young people favours the molar region and is unrelated to the vitality of neighbouring teeth The initiating trauma is thought to lead

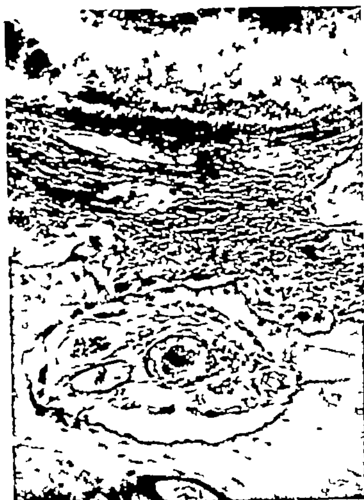


FIG. 201
A photomicrograph depicting the area in X Figure 200 at a higher magnification There is new bone forming in the fibrous lining to the cavity side by side with the osteoclastic resorption of the outer plate of bone Haematoxylin and eosin ×130

a haematoma and localized necrosis of marrow which for reasons that are not understood is not followed by the normal reparative process. Osteoclastic resorption of the adjacent trabeculae proceeds surrounding cavity lined by granulation tissues and areas of haemorrhage (Figs. 200 201). At operation there is no dissectible sac, and the mandibular nerves and vessels may be lying free in the cavity

4. BONE DYSTROPHY

Paget's Disease is a localized bone disease of patchy distribution affecting the whole or part of a bone, or many bones It principally affects older people, being uncommon under the age of forty The age incidence gives support to Jaffe's view (1933), that the cause of Paget's disease may be a breakdown of the normal mechanism of creeping replacement to which bone is constantly subject While the weight-bearing

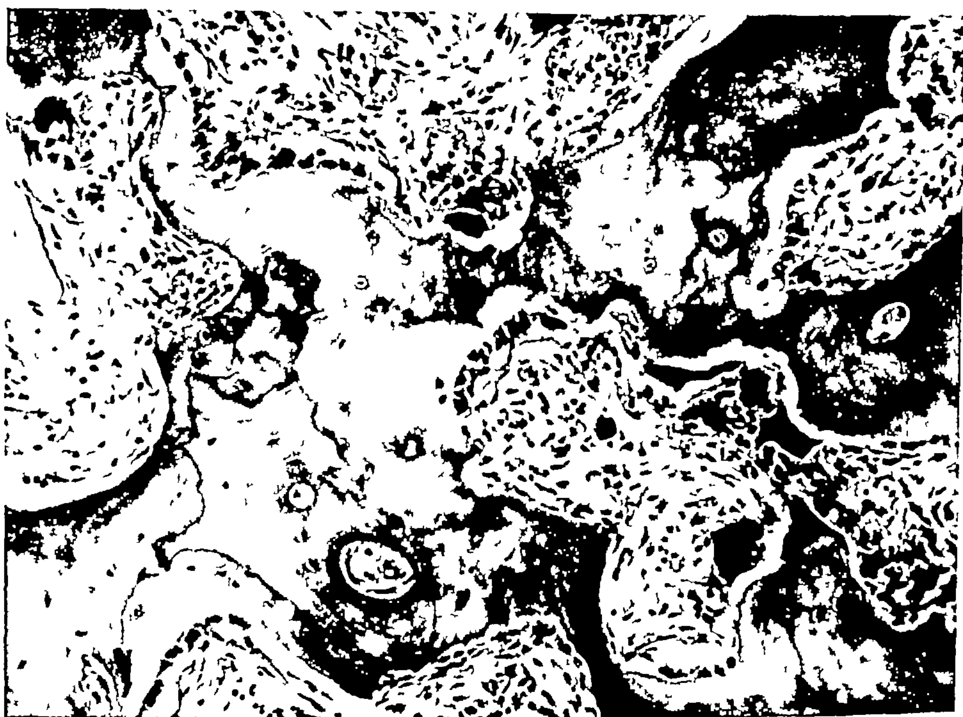


FIG 202

Paget's disease Irregular trabeculae of bone with a mosaic pattern, marked osteoclastic and osteoblastic activity, and fibrosis of the marrow Haematoxylin and eosin ×110

bones such as the pelvis and spine are commonly involved, the skull and jaws, particularly the upper, may be affected too, leading to thickening and deformity

The characteristic picture results from an increased turnover of bone, in which more bone is formed than resorbed The original bone is completely resorbed and replaced by irregular masses of bone consisting of small fragments of either lamellar or coarse-fibred woven bone, each fragment being demarcated by several lines Osteoblastic and osteoclastic activity proceed side by side, and the bone is given a typical 'mosaic pattern' by the repeated apposition and resorption (Fig 202)

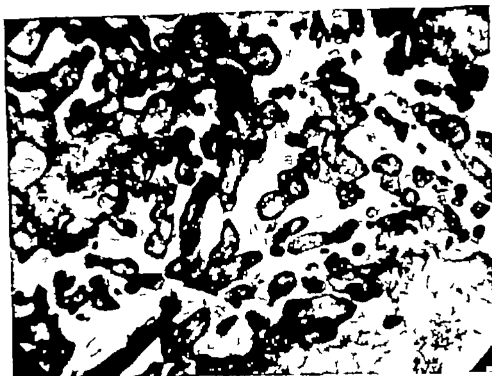


FIG. 203

Paget's disease of maxilla Small sclerotic nodules of bone in fibrous tissue Haematoxylin and eosin ×55 (*Ann Roy Coll Surg Engl* 1956)

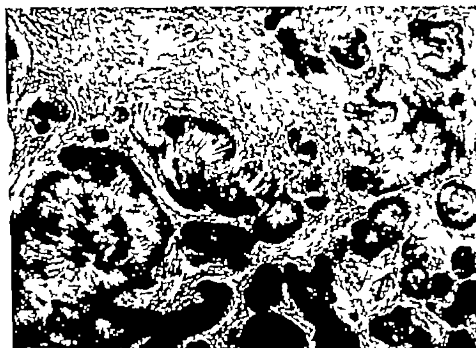


FIG. 204

Paget's disease of maxilla. The small nodules are fused together by woven bone having a fibrillar structure at right angles to their surfaces. Haematoxylin and eosin. ×120 (*Ann Roy Coll Surg Engl* 1956)



FIG 205
Paget's disease of the maxilla This shows the transition from the small sand-like calcified nodules just beneath the alveolar crest, to the larger irregular shaped masses in the deeper tissues Haematoxylin and eosin ×6
(*Ann Roy Coll Surg Engl* 1956)

The fibrosis of the marrow is secondary to the resorptive process Sometimes following osteoclastic activity small dense sclerotic nodules of bone that rarely enclose cells are formed (Fig 203) The smaller nodules become fused together into larger irregular dense masses (Fig 204) some of which become united with the cementum of adjacent teeth (Fig 205)

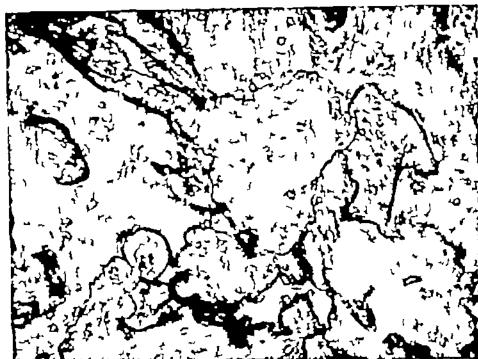


FIG 206

Paget's disease of maxilla Mosaic pattern in cementum Haematoxylin and eosin ×120 (*Ann Roy Coll Surg Engl* 1956)

The teeth in the affected area may show an irregular hypercementosis This new cementum is cellular and sometimes shows curvilinear markings of remodelling even to the extent of forming a mosaic pattern similar to that seen in the adjacent bone (Fig. 206)

5. NEOPLASMS

The Ossifying Fibroma is a benign neoplasm of fibrous tissue directly associated with the skeleton It is characterized by scattered areas of acellular calcification that do not occur in the normal formation of membrane bone, and it is subperiosteal rather than central in origin It is encapsulated The microscopical appearance shows sheets of fibroblasts arranged in a whorled pattern and there is some periosteal lamellar bone at the periphery Scattered throughout the fibrous tissue are small irregularly shaped and globular calcified masses that stain

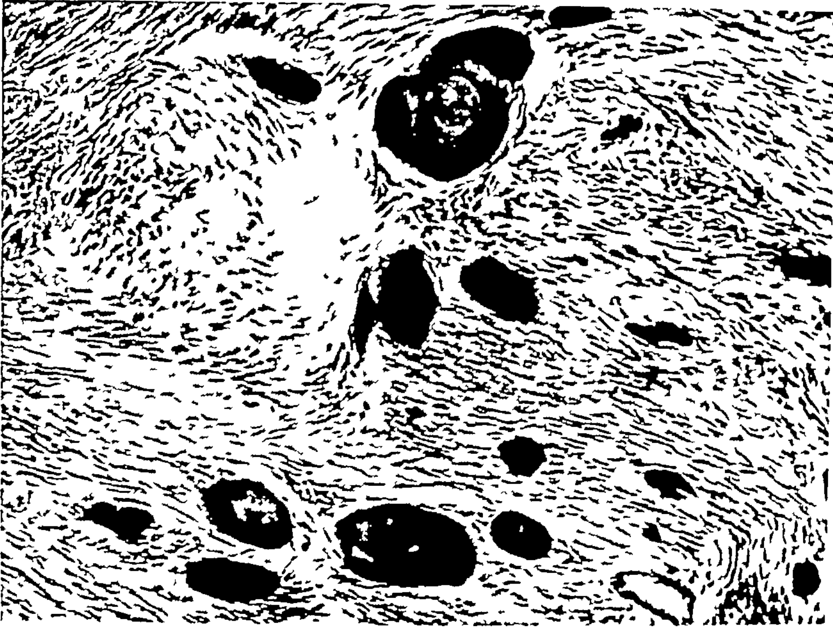


FIG 207

Ossifying fibroma of the maxilla Non-cellular haematoxophil calcific masses are scattered in the whorled pattern of fibrous tissue Haematoxylin and eosin ×60 (*Brit dent J* 1957)



FIG 208

Ossifying fibroma of mandible Irregular trabeculae of coarse-fibred calcified matrix devoid of osteocytes Haematoxylin and eosin ×120 (*Brit dent J* 1957)

deep blue with haematoxylin especially at their periphery (Fig. 207) In other fields the calcification occurs in a hyaline substance that stains pink with eosin and can be seen surrounding calcified deposits Only very rarely indeed is a cell included and the masses fuse together to form irregular shaped areas that hardly deserve the title of trabeculae (Fig 208)

The Fibro-osteoma is a benign tumour of osteogenic fibrous tissue that undergoes almost the full range of the formation of bone in



FIG 209

Fibro-osteoma of maxilla Mass of woven bone being resorbed and replaced by lamellar bone Haematoxylin and eosin × 120 (*Brit dent J* 1957)

membrane It tends to be subperiosteal rather than central and invokes in the adjacent bone a reactive sclerotic margin There appears to be a slowing up of its growth with increasing age

Bone formation in a fibrous stroma is the essential feature of the microscopical appearance The fibrous tissue is less vascular and more collagenous and lacks the whorled pattern of the ossifying fibroma. The full range of bone formation from osteoid and woven bone to lamellar bone may be observed (Fig 209) Remodelling goes on until the older lesion is almost a solid mass of bone with just a rim of neoplastic fibrous tissue at the periphery (Fig 210) The non-cellular haematoxophil calcified spherules such as seen in the ossifying fibroma are rarely observed.

The Compact and Cancellous Osteoma. The osteoma is a benign neoplasm of bone that grows by the continuous formation of lamellar bone, and it is not a stage in the development of a fibro-osteoma in which the bone formation is secondary rather than primary.



FIG 210
Fibro-osteoma of mandible Large mass of bone that is a mixture of lamellar and woven bone enclosing a few fibrous marrow spaces Haematoxylin and eosin ×60 (*Brit dent J* 1957)

In microscopical appearance the compact osteoma shows on its periosteal surface layers of lamellar bone with little evidence of remodelling, rather like an onion (Fig 211) In the deeper part of the lesion there may be a coarse mosaic pattern, but there is no organization of bone around Haversian systems

Figure 212 shows an unusually active osteoma surrounded by a rim of fibrous tissue in which coarse-fibred woven bone formation precedes the lamellar bone formation In contrast it is not possible to distinguish histologically the inactive osteoma illustrated in Figure 211 from a torus or from simple hyperplasia of bone

The cancellous osteoma consists of trabeculae of lamellar bone surrounding fatty or fibrous marrow spaces The fibrous marrow reverts to fatty marrow when there is little osteoclastic or osteoblastic activity

The Chondroma and Chondrosarcoma. These cartilaginous tumours are extremely rare in the jaws and are not easy to distinguish one from another They are unpredictable in their behaviour and one that clinically and microscopically appears to be benign may later assume sarcomatous qualities Lichtenstein and Jaffe (1943) suggest that a tumour of cartilage is malignant if (*a*) many cells with plump nuclei are to be seen (*b*) more



Fig 211
Compact osteoma of mandible. Layers of lamellar bone with little evidence of remodelling Haematoxylin and eosin. × 70

than an occasional cell has two such nuclei (*c*) there are giant cartilage cells with large single or multiple nuclei or with clumps of chromatin (Fig. 213)

The Osteogenic Sarcoma. The osteogenic sarcoma is fortunately a rare tumour of the jaws for it is very malignant with early metastases to the lungs.



FIG 212
Compact osteoma of maxilla Woven bone formation at the active margin of the osteoma Haematoxylin and eosin × 130

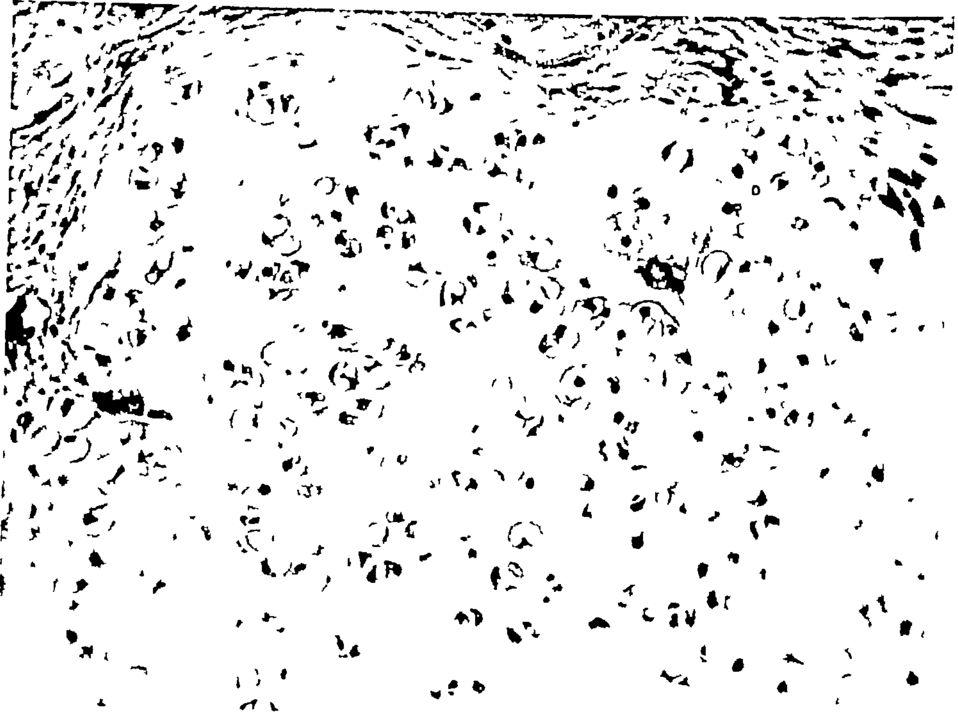


FIG 213
Chondrosarcoma of maxilla with pleomorphic cartilage cells Haematoxylin and eosin × 140 (From a specimen kindly lent by Professor R B Lucas)

The microscopical appearance is characterized by great cellular pleomorphism with an admixture of spindle cells multinucleated giant

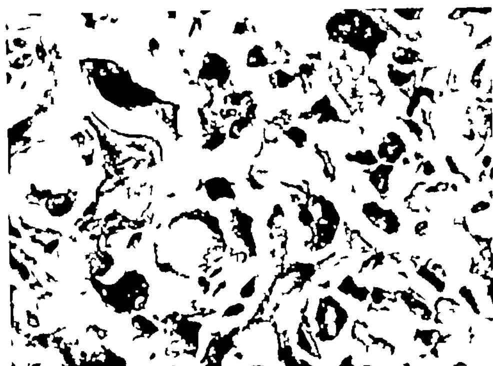


FIG. 214

Osteogenic sarcoma of mandible with multinucleated osteoblasts lining the immature bone Haematoxylin and eosin ×410 (From a specimen kindly lent by Professor R. B Lucas.)

cells, coarse fibred woven bone and sometimes cartilage (Fig 214) In fact this tumour has perhaps the widest range of histological pattern of any tumour

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